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Department of Internal Medicine №2

METHODOLOGICAL MATERIALS ON GASTROENTEROLOGY IN THE COURSE OF FACULTY THERAPY

main professional educational program of higher education - specialty program in the specialty 31.05.01 General Medicine

Vladikavkaz

Methodological materials are intended for teaching 4th year students (7.8 semesters) of the Faculty of General Medicine. FSBEI HE NOSMA of the Ministry of Health of the Russian Federation on the discipline "Faculty therapy".

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CONTENT:

CHRONIC GASTRITIS

Chronic gastritis (CG)- chronic inflammation of the gastric mucosa, manifested by cellular infiltration, impaired regeneration and, as a result, atrophy of the glandular epithelium, intestinal metaplasia, disorder of the secretory, motor and often endocrine function of the stomach. Simply put, chronic gastritis is inflammation of the stomach in response to injury.

Etiology and pathogenesis

There are 3 groups of causes that contribute to the onset of chronic hepatitis:

Infectious:the most common cause of chronic hepatitis is Helicobacter pylori. Much less often - this is the infection of the mucous membrane with other microbes, viruses, fungi, parasites. In 80% of cases, chronic hepatitis is caused by HP. This microbe is a flagellate gram-negative microaerophilic bacterium with a spiral (helicos) shape. Coccal forms are also distinguished, which are probably necessary for persistence in an unfavorable environment. The pathogenicity of HP is associated with its mobility, the release of enzymes, among which the most important is urease. Urease ensures the introduction of HP into the mucous membrane (CO), participates in its further damage, promotes the activation of monocytes and polynuclear cells with the release of pro-inflammatory cytokines.

Once in the stomach, HP is embedded in the mucus layer that covers the antrum and is located on the surface of epithelial cells. The primary mechanism of damage is the infiltration of the mucous membrane by neutrophils, then HP contributes to the lymphocytic and plasma cell infiltration of CO. Thus, HP destroys gastric CO, contributes to the development of atrophy.

Non-infectious causes

1. Immunological factors. They lead to the formation of autoimmune gastritis. In these patients, autoantibodies are found in the blood against parietal (parietal) cells of the stomach that produce hydrochloric acid and internal Castle factor. The latter is necessary for the absorption of vitamin B12.

Autoimmune gastritis is manifested by widespread CO atrophy in the fundus and a marked decrease in acid production in the stomach. A number of patients simultaneously develop B-12 deficiency anemia.

Atrophic gastritis is determined by the following features: 1) antibodies to parietal cells (in some patients and to the internal Castle factor), 2) atrophy of the body of the stomach. HCA is often called genetically determined, and the atrophy that forms with it is called primary. Often, such gastritis is combined with Hashimoto's thyroiditis, type 1 diabetes mellitus, vitiligo, and other autoimmune diseases.

2. Prolonged exposure to aspirin and other non-steroidal anti-inflammatory drugs, alcohol or chemicals, bile reflux into the stump of the resected stomach lead to the formation of type C gastritis (chemically - toxicly induced).

3. Nutrition and environmental factors. In the description of the etiology of gastritis since 1997. re-name the dietary habits that were absent as causal factors since 1990. The focus on HP has called into question the role of eating disorders in the origin of hCG. Without stating the primary etiological role of diet disorders, each clinician can talk about the essential role of regular balanced nutrition in maintaining clinical remission of the

disease against the background of etiotropic therapy.

Pathogenesis

- Infectious inflammation: HP, other microbes.
- Autoimmune, genetically determined inflammation.
- Medicinal and chemical damage to cutting fluid.
- Violation of the motor function of the stomach.
- Increased aggressive potential of gastric juice in some patients.
- Disturbance of gastric digestion with atrophy of the fundic coolant.

Morphology

In the clinical diagnosis, first of all, it is required to reflect the morphological form of gastritis, the localization of these changes in the stomach and the etiology, if known.

By localization and length, hCG is distinguished:

- antrum (antrum)
- fundus (body of the stomach)
- pangastritis (total).

The depth of the lesion is distinguished:

1. Superficial, or non-atrophic gastritis: lymph - plasmacytic infiltration of CO. This is the most common form of chronic hepatitis. The prognosis of superficial gastritis is favorable.

2. Atrophic gastritis. This is a progressive process. It begins as a superficial inflammation and, through the gradual loss of the gastric glands, ends with a total atrophy of the coolant. The main cells that produce pepsin, parietal cells disappear, cells that produce mucus appear. Atrophic gastritis can be diffuse (antral, fundus, total) or multifocal.

Isolated atrophic gastritis of the body with little altered mucous membrane of the antrum is usually autoimmune, genetically determined. Atrophic gastritis in the antrum in the overwhelming majority of cases is associated with Helicobacter pylori etiology, since HP is tropean to the mucous membrane of this particular part of the stomach. Pangastritis, as a rule, is of Helicobacter nature. The main role in the progression of chronic hepatitis is played by epithelial proliferation, which prevails over differentiation processes. Cells rejuvenate, but do not have time to acquire mature properties.

3. Metaplasia of the coolant. Metaplasia can be pseudopyloric and intestinal. With intestinal metaplasia, all cellular elements characteristic of the intestinal epithelium are determined in the areas of the coolant: enterocytes, Paneth cells.

The gastric ridges begin to resemble villi, and the pits begin to resemble crypts of the small intestine. The appearance of all the properties of the small intestinal epithelium was previously called enterolization, and now it is complete intestinal or small intestinal metaplasia. In cases where the metaplastic cells more resemble the colonic epithelium, they speak of incomplete metaplasia.

Complete metaplasia is most often found. Incomplete metaplasia or small bowel metaplasia is a more unfavorable prognostic factor and is considered a precancerous change.

4. Dysplasia. This is a deviation from the normal structure of the cells of the tissue complex in the direction of the neoplastic process. The concept of dysplasia has been proposed to denote precancerous changes and the formation of high-risk groups. Dysplasia is spoken of in the presence of cellular atypia, a change in the shape of the gastric glands. Patients with dysplasia require the supervision of a therapist, endoscopist, and morphologist.

Classification

The first etiological classification was proposed in 1973. In the classification of Dixon (1989), chronic gastritis A, B, C are distinguished, and these letters are not only the equivalent of a serial number, but also the beginning of a word characterizing the type of gastritis. A - autoimmune, B - bacterial, C - chemically mediated.

The first generally accepted international classification of CG is the Sydney system. This classification was adopted in 1990 in Sydney. It regulates endoscopic and histological protocols. In 1996. this classification has been modified.

According to ICD 10 - 10, there are:
K29 Gastritis and duodenitis
K29.0 Acute hemorrhagic gastritis
K29.1 Other acute gastritis
K29.2 Alcoholic gastritis
K29.3 Chronic superficial gastritis
K29.4 Chronic atrophic gastritis
K29.5 Chronic gastritis
unspecified
antral
fundamental
K29.6 Other gastritis
hypertrophic giant gastritis
granulomatous gastritis
Menetrie's disease
K29.7 Gastritis, unspecified

Diagnosis formulation. Examples.

1) Chronic HP - associated superficial pangastritis, f. exacerbation.

2) Chronic active HR - associated antral gastritis with focal atrophy of the glands and intestinal metaplasia

3) Chronic HP - associated superficial gastritis with atrophy and erosions in the antrum of the stomach.

4) Autoimmune chronic gastritis with a predominance of severe atrophy in the fundus of the stomach, exacerbation.

5) Reactive (chemical) antral gastritis with erosions associated with the intake of indomethacin.

Clinic

Pain- the most common complaint of any gastroenterological patient, as well as a patient with chronic hepatitis. It is necessary to answer the following questions: 1) Where is the pain localized? It can be assumed that the localization of pain is 60% of the diagnosis. To determine the localization, the patient must be asked to stand up and show the place where he feels pain. Pain in chronic hepatitis is usually felt in the center of the epigastrium (fundic gastritis, pangastritis), but its localization is possible in the pylorobulbar zone (antral gastritis) 2) Is the pain associated with food intake or other external causes? With fundic gastritis or pangastritis, pain usually occurs 10 to 20 minutes after eating (early pain). It has an aching character, localized under the xiphoid process, or throughout the epigastrium. Palpation of the abdomen in many cases reveals moderate diffuse soreness in the epigastric region.

Gastric dyspepsia syndrome (a complex of sensations in the form of nausea, heaviness in the epigastrium, a feeling of overflow, discomfort, intensifying, as a rule, after eating and localized in the epigastrium. Vomiting is of an episodic nature and is associated with eating disorders (a long break in eating, taking a significant amount of drugs, smoking) , brings relief.Stubborn, repeated vomiting is a reason to look for more serious diseases (chronic pancreatitis, peptic ulcer, cancer, etc.)

*Symptom of insufficient function of cardiac pulp and gastroesophageal reflux:*belching and heartburn. Taste in the mouth: metallic, sour and bitter sensation. The role of duodenogastric and gastroesophageal reflux, biliary dyskinesia is indicated. Appetite and weight usually do not change with chronic hepatitis. State of the central nervous system: In a patient with chronic hepatitis, the doctor often encounters the monotony of the pain syndrome, including its constant nature, which does not depend on any external phenomena, and the absence of any positive effect from taking medications, as well as often excessive and unusual symptoms , colorful and unusual descriptions of symptoms. These situations indicate that the patient has both an underlying disease and psychosomatic pathology, hypochondria and depression. These patients report feeling anxious, depressed mood, and sleep disturbance. Possible elements of cancerophobia in the mood and behavior of the patient. Complaints are presented, the intensity of which is in clear discrepancy with the severity of chronic hepatitis. This should be taken into account in the treatment.

The history of the disease in chronic gastritis differs in duration and is usually at least two years.

Physical examination: external signs of the disease are often absent, the general condition of patients is satisfactory. Rarely, there is a lack of weight, pallor of the skin, a symptom of hypovitaminosis (cracks in the corners of the mouth, bleeding gums, hyperkeratosis, brittle nails, hair loss), which can be attributed to chronic hepatitis only with diffuse and significantly pronounced atrophy of the CO of the body or the entire coolant. The tongue is often coated with a white and yellow-white coating with dental imprints on the lateral surface. The abdomen is soft on palpation, sometimes slightly swollen. In patients with diffuse chronic hepatitis, moderate diffuse soreness in the epigastric region is determined, with antral gastritis, local soreness in the pyloric-duodenal zone.

Survey

Obligatory laboratory tests

Once:

- general blood analysis
- fecal occult blood test

- histological examination of biopsy
- cytological examination of biopsy
- two tests for HP
- total protein and protein fractions
- general urine analysis

Mandatory instrumental studies

Once: Esophagogastroduodenoscopy with targeted biopsy and brush cytological examination. Ultrasound of the liver, biliary tract and pancreas Additional studies and consultations of specialists are carried out depending on the manifestations of the underlying disease and the alleged concomitant diseases

Treatment

Diet therapy. 1. Regularity of food. Rules: meals should be regular, 3-4 times at the same hours. After eating, you can not go to bed or work in an incline for 40 minutes.

Food should be mechanically, chemically and thermally gentle. It is required to exclude spicy, fatty, fried foods, spices, smoked meats, black bread, carbonated drinks, mushrooms, bacon, marinades from the diet for 2-3 weeks. Raw vegetables and fruits are excluded for 5-7 days.

Drug therapy.

Symptomatic therapy is prescribed from the moment the patient applies for medical help.

Antacids. Therapeutic effects: a) neutralization of hydrochloric acid, b) adsorbing, enveloping effect, c) pain reduction due to the implementation of the first two effects, d) cytoprotective effect.

In the mode of using antacids for chronic hepatitis (Z r per day 7-14 days), they are devoid of side effects and do not cause changes in acid-base and mineral balance. In the absence of data on the etiology of chronic hepatitis, antacids, together with drugs that affect motility, are actually the main method of treating chronic hepatitis. Antacids include Almagel, Phosphalugel, Gastal, Gestid, Rennie, Almazilat. Gel antacids act faster than tablets, but the latter are more convenient to use. The standard regimen for taking an antacid: 1t 3 r. Per day for 30-6 Omin before meals or 1-1.5 hours after meals (depending on the time of the onset of pain) 7-14 days.

Antacid is a basic drug for relieving symptoms of exacerbation. If pain persists against this background, a drug that affects gastric motility may be added to it. There are two groups of drugs: antispasmodics and prokinetics (activating gastric motility). When the prevalence in the structure of complaints is not so much pain as dyspeptic syndrome with nausea, heaviness, belching, stomach discomfort, antacid is usually combined with a prokinetic.

Prokinetics activate the peristalsis of the stomach, duodenum, increase the tone of the muscles of the stomach, the tone of the cardia, eliminating belching, nausea, vomiting, hiccups. One of the above drugs is prescribed:

Metoclopramide (raglan, cerucal), domperidone (motilium), cisapride (coordinax). Assign 1t Zr. per day for 20 min. before meals 2 weeks.

Antispasmodics. If pain prevails in the HCG clinic, it is primarily associated with spasm. In this case, antispasmodics are used in combination with an antacid. Myotropic antispasmodics may be prescribed: no-shpa, halidor, 1-2 tabs each. Zr. per day, these drugs are rarely used now. Most often, M-choligolytics are prescribed: metacin at 2-4 mg Zr. a

day 15 minutes before meals, bacarbon, pirenzepine (gastrocepin, gastril, pyrene, gastrozem) 25-50mg 2p. a day 15-30 minutes before meals.

Antisecretory drugs are drugs that reduce the production of somatic acid by the parietal cells of the stomach. They are represented by two chemical groups: H2 blockers and proton pump blockers.

H2 receptor blockers:

- have an antisecretory effect, suppress the basal and stimulated by histamine, gastrin, the intake of ordinary food, the nocturnal secretion of hydrochloric acid.
- reduce the release of pepsin
- increase the production of gastric mucus
- improve microcirculation in the mucous membrane of the stomach and duodenum
- normalize gastroduodenal motility.
- increase the production of prostaglandin E, which mediates the cytoprotective effect, i.e. drugs in this group promote the healing of ulcers. Ranitidine (Gastak, Zantac, Ranisan) is prescribed at 150 mg. 2p per day for 1 week, then 1 tab. in the evening - 1 week, famotidine (lesedil, gastrosidine) 40 mg. in the evening 1 week, 20 mg. - in the evening 1 week.

Proton pump inhibitors omeprazole (omez, romesec) is prescribed 20mg 1-2 p. per day 2-3 weeks. In chronic hepatitis, proton pump inhibitors are given less frequently than H2 blockers.

The synthetic analogue of prostaglandin E1 is misoprostol (cytotec). It has a cytoprotective effect associated with an increase in the production of mucus in the stomach and an increase in the secretion of bicarbonate of the coolant, improves blood flow.

Misoprostol is the main drug for the treatment of HCG associated with the use of NSAIDs, manifested by the presence of erosions. The positive effect that prevents the implementation of the action of NSAIDs is associated with the fact that misoprostol increases the content of cyclooxygenase-1 (COX-1) in the coolant. The formation of erosion is associated with a decrease in this particular enzyme.

Sucralfate (Venter) is an aluminum salt of sulfated sucrose. Accelerates the healing of ulcerative fluid defects by forming a chemical complex - a protective barrier on the surface of an erosive or ulcerative defect, prevents the action of hydrochloric acid, pepsin, bile. It is insoluble in water, therefore it is adsorbed in a minimal amount and retains its local effect. Prescribe 0.5 Z p. per day 1 hour before or 1 hour after meals and 1 tab. at night 2 weeks.

Psychotropic drugs are prescribed for patients with concomitant neuroses: astheno-neurotic, astheno-depressive and astheno-hypochondriacal. Anxiolytics (anti-anxiety), hypnotics, antidepressants are prescribed. If the effect is insufficient, a psychiatrist's consultation is indicated.

Autoimmune gastritis treatment.

The total number of patients with ACG with widespread atrophy of the gastric corpus or total atrophy of the mucous membrane of CO is estimated at one percent of the population.

Due to the lack of a pathogenetically justified remedy, diet and prokinetics

(cisapride, cerucal, domperidone) are very important. It is possible to use sucralfate for 2-3 weeks. In the presence of B12 deficiency anemia, oxycobalamin or cyanocobalamin is prescribed 1000 mcg IM for 6 days, then in the same dose for 1 month the drug is administered once a week, then for life once every 2 months.

Hospitalization is indicated for patients with pronounced manifestations of an exacerbation of the disease, with erosive lesions of the gastric mucosa (risk of bleeding), as well as with difficulties in the differential diagnosis of chronic hepatitis and gastric cancer.

Prophylaxis

Active detection of hCG is important to prevent its progression. The earliest possible detection of helicobacteriosis and eradication of HP, that is, treatment according to the above rules, plays an important role in slowing the progression of morphological changes in the coolant. Monitoring persons regularly taking NSAIDs is an important measure for the prevention of type C gastritis. Due to the high incidence of stomach cancer, it is advisable to regularly monitor patients with underlying diseases such as chronic hepatitis and gastric polyps. For all forms of chronic hepatitis, regular courses of physiotherapy and annual spa treatments are beneficial. The main resorts for the treatment of diseases of the digestive system: Arzni, Borjomi, Essentuki, Goryachy Klyuch, Druskininkai, Zheleznovodsk, Morshin, Pyatigorsk, Staraya Russa, Sestroretsk, Pyatigorsk.

GASTRIC and duodenal ulcer

In different age periods, gastroduodenal ulcer occurs in 4-5% of the population. According to the Ministry of Health of the Russian Federation, about 3.5 million people are registered with nuclear medicine, of which every 10 was operated on. About 6,000 people die from complications of ulcer in our country, and 6,500 people die in the USA. Deaths from ulcers in the UK outnumber deaths from injuries in car accidents, breast and cervical cancer.

Peptic ulcer - a chronic disease with a recurrent course and a tendency to progression with a morphological equivalent in the form of a defect in the mucous and submucous layer with an outcome in the connective tissue scar.

Etiological factors in the development of ulcer:

- neuro-emotional stress;
- hereditary predisposition;
- hyperplasia of parietal cells;
- violation of the synthesis of factors of protection of the mucosa;
- susceptibility of the mucous membrane to HP;
- hypervagotonia, hypergastrinemia;
- insufficiency of pancreatic secretion of bicarbonate;
- other chronic diseases;
- violation of the diet;
- smoking, alcohol abuse, medication.

Helicobacter pylori

is the cause of the development of duodenal ulcer (DU) in 80 - 90% of patients, gastric ulcer (PU) in 60 - 70% of patients. Infection with HP during adolescence increases the lifetime risk of coronary heart disease.

Factors affecting the prevalence of H. pylori:
Economic;
Social:

- education, profession;
- marriage and family composition;
- religious affiliation;
- isolated populations.

Racial;
Age-related;
Waste products Nr:

- violate the integrity of the mucous membrane;
- contribute to the development of inflammation in it;
- potentiate the processes of dysplasia and metaplasia;
- initiate immunological reactions;
- increase the rate of secretion of hydrochloric acid (HCl).

The pathogenesis of ulcer is not fully understood. It is based on imbalances between the factors of aggression and defense.

In the pathogenesis of DU, the predominant role is played by: acid-peptic factor, accelerated evacuation from the stomach, acid stasis.

In ulcer of the antrum of the stomach - duodeno-gastric reflux, antral stasis, high secretion of HCl.

In case of mediogastric ulcer, there is an insufficient mucous barrier, impaired vascularization, a lack of production of prostaglandins and Ig.

DIAGNOSTICS OF THE PURPOSE DISEASE

MAIN SYNDROMES:

Pain syndrome occurs as a result of exposure to acid-peptic factor, spasm of the pyloric-duodenal zone, increased pressure in the stomach and duodenum, periulcerous inflammation, irritation of visceral sympathetic fibers.

Dyspepsia syndrome... A feeling of pain or discomfort (heaviness, overflow, early satiety), localized in the epigastric region (closer to the midline), occurs when motor and evacuation functions are impaired; unpleasant taste in the mouth; belching, often empty, airy, less often food; heartburn is sometimes excruciating, as the equivalent of pain; nausea; vomiting - more often at the height of a painful attack without prior nausea, relieves pain. Often, patients cause it artificially.

Astheno-vegetative syndrome.

Complex manifestation of disorders of the autonomic nervous system:

- anxiety, hypochondria, irritability, weakness; egocentrism, demonstrativeness.

Signs of vegetative dystonia:

- arterial hypotension, pulse lability;
- acrocyanosis, cold hands, hyperhidrosis;

- mitral valve prolapse;
- early repolarization syndrome on the ECG.

Time of onset of pain syndrome in ulcer:

With an ulcer of the pyloric part of the stomach and the duodenal bulb, the pain is late, 1.5 - 2 hours after eating, nocturnal, hungry, stopped by antacids and food.

With an ulcer of the body and the cardiac part of the stomach, the pain is early, 1 / 2-1 hour after eating, stops after emptying the stomach.

With a postbulbar ulcer, the pain is intense, pulsating, 3 to 4 hours after eating and is often stopped only by drugs.

Localization of pain in ulcer:

With ulcers of lesser curvature - in the epigastrium to the right of the midline.

With ulcers of the cardiac department - in the zone of the xiphoid process.

With pylorobulbar ulcers - to the right of the midline and 5-7 cm above the navel.

The appearance of radiating pain usually indicates a complication of ulcer:

- penetration into neighboring organs;
- the development of perivisceritis, solar syndrome;
- the presence of concomitant diseases.

Peculiarities of ulcer in children:

- ulcerative defects are more often localized in the duodenum (90%);
- pain is uncertain and localized;
- diffuse pain in the navel in preschoolers;
- pain monthly or slightly at large intervals;
- dyspepsia and atypical clinical course are common.

Peculiarities of PU in the elderly:

- Peptic ulcer disease is a continuation of the disease from a young age:
- characterized by a clear seasonality;
- has a progressive course;
- the rhythm of the pain syndrome is disturbed;
- the predominance of dyspepsia;
- latent bleeding is detected in almost 50% of individuals;
- large ulcerative defect;
- slow rates of reparation;
- frequent presence of concomitant pathology (atherosclerosis, etc.).

Objective symptoms of ulcer:

S. Troitsky - seasonal and food frequency of pain.

S. Openkhovsky - pain in the area of spinous processes of the VII-X thoracic vertebrae.

S. Boasa - soreness on both sides of the spine at the level of the X-XII thoracic vertebrae.

S. Herbst - soreness at the level of the transverse processes of the III lumbar vertebra.

S. Grekov - a slowdown in the pulse in the first hours after the perforation of the

ulcer.

S. Bergman - disappearance of pain in the abdomen following the onset of gastrointestinal bleeding.

S. Schlizinger - with pyloric ulcers - transient displacement of the navel towards the lesion when straining.

S-m Laenek - pain when pressing the epigastric region with the abdomen pulled in.

In order to increase the effectiveness of treatment of patients with diseases of the digestive system by systematizing modern approaches to the tactics of managing patients and unifying the optimal modes of the treatment and diagnostic process, on April 17, 1998, the Ministry of Health of the Russian Federation issued Order No. 125

"On the standards of diagnosis and treatment of patients with diseases of the digestive system", mandatory for use in all medical institutions in Russia, including in the work of general practitioners and outpatients.

The scope of examinations of patients with ulcer:

- General blood analysis;
- Blood group, Rh factor;
- Fecal occult blood test;
- Esophagogastroduodenoscopy;
- Biopsy examination;
- Study of acid production in the stomach;
- Ultrasound of the digestive system;
- X-ray examination of the gastroduodenal zone.

Methods for diagnosing helicobacteriosis:

- gastroscopic biopsy;
- respiratory analytical (using carbon isotopes)
- serological.

Endoscopic markers of helicobacteriosis:

- antral gastritis, including erosive;
- duodenitis, including erosive;
- ulcer of the duodenal bulb;
- a combination of the above features.

Nuclear weapons classification according to ICD-10:

- K.25 Stomach ulcer
- K 26 Duodenal ulcer
- K 27 Peptic ulcer, unspecified
- K 28 Gastrojejunal ulcer

Classification of ulcer according to the severity of the course:

- rarely recurrent (exacerbation 1 time in 5 years);
- mild course (exacerbation 1 time in 1-3 years);
- moderate (exacerbation 2 times a year);
- severe (exacerbation 3-4 times a year);

- continuously recurrent (exacerbation 4 or more times a year, there are no periods of remission);
- latent (25% of all ulcers detected and confirmed by preventive endoscopic examinations of patients).

Complications of peptic ulcer:

- bleeding;
- perforation;
- penetration;
- deformity and stenosis;
- malignancy;
- perivisceritis;
- reactive hepatitis;
- reactive pancreatitis.

An example of a diagnosis formulation:

- duodenal ulcer;
- bulb ulcer 0.5-0.6 cm;
- moderate course: exacerbation phase;
- erosive duodenitis. Hp +.
- increased acid-forming function of the stomach.
- cicatricial deformity of the duodenum.

The role of proton pump inhibitors (PPIs) in the treatment of ulcer:

- Suppress acid formation by 100%
- Quickly relieve the symptoms of ulcer and promote the rapid scarring of ulcerative defects
- Reduced relapses and complications
- There is no resistance to the action of PPIs
- They increase the stability and duration of antibiotic action during eradication therapy.

Peptic ulcer disease without H. pylori eradication will remain "chronic recurrent suffering", therefore, anti-Helicobacter pylori therapy must be carried out even in the remission phase:

triple scheme of the first line 7 (10) days:

PPI (OMITOX) 20 mg x 2 r / d
+ Clarithromycin 500 mg x 2 r / d
+ Amoxicillin 1000 mg x 2 r / d or Metronidazole 500 mg x 2 r / d
the effectiveness of the course of therapy is 90%;

second line quadrotherapy 10 (14) days:

PPI (OMITOX) 20 mg x 2 r / d
+ Bismuth preparations 120 mg x 4 r / d
+ Tetracycline 500 mg x 4 r / d or Metronidazole 250 mg x 4 r / d
the effectiveness of the course of therapy is 96%.

Ulcers with successful anti-Helicobacter pylori therapy scar faster and better than with traditional antiulcer treatment

Aspects to consider when choosing an antibacterial drug:

- Safety
- Good tolerance
- Ease of use
- Optimal frequency of reception
- Good organoleptic properties
- Pharmaco-economic aspects

The activity of macrolides against H. pylori is of primary clinical significance for GE and GP.

Macrolides are among the least toxic antibiotics

The effectiveness of Azithromycin in the eradication of Hp as part of combination therapy today is 86% with a three-day treatment regimen of 1 g per day

Rules for conducting anti-Helicobacter pylori therapy:

- You should not repeat a treatment regimen that was ineffective at the first treatment;
- Failure means acquired Hp-resistance to one of the components of the treatment regimen.
- The appearance of Hp a year later in the patient's body after the course of eradication should be regarded as a relapse of the infection and more effective treatment regimens should be prescribed.

At the end of eradication therapy, you should continue taking the used antisecretory drug in a half dose for another 7-10 days or switch to taking an antacid (1.5-2 hours after eating and before bedtime).

Purpose of peptic ulcer treatment:

- ulcer healing
- achieving stable remission

Tasks of ulcer treatment:

- relief of emergency conditions;
- elimination of clinical symptoms;
- eradication of helicobacteriosis;
- healing of ulcers ("pink scar");
- achieving a stable clinical remission ("white scar").

Indications for hospitalization in a surgical hospital:

- perforated ulcer;
- bleeding ulcer;
- decompensated pyloric stenosis;
- penetrating ulcers.

Indications for planned hospitalization in the gastroenterology or internal

medicine department

- The presence of social indications:
- dormitory accommodation;
- poor living conditions;
- the inability to organize a full-fledged dietary food at home.

The organization of treatment of patients with uncomplicated form of ulcer in day hospitals is widely practiced.

For the prevention of complications of ulcer, two types of therapy are recommended:

1. "Continuous" (for months and even years) maintenance therapy with an antisecretory drug in a half dose, for example: you should take 20 mg of OMITOX daily in the evening.

2. "On demand".

Indications for "continuous" prophylaxis of ulcer:

- ineffectiveness of the performed HP eradication;
- the need for constant use of NSAIDs;
- concomitant erosive and ulcerative reflux esophagitis;
- the presence of complications of ulcer;
- the age of patients is over 60 years old;
- annual relapses of ulcer, despite adequate course therapy.

Indications for prophylaxis of ulcer "on demand":

Manifestation of symptoms of ulcer after successful preliminary eradication of Hp in a patient who responds adequately to the disease.

When symptoms of ulcer appear, it is advisable to take one of the antisecretory drugs in a full daily dose for 3 days, then in a half dose for 3 weeks (for example: OMITOX 40 mg - 3 days, then 20 mg)

PUD prevention also includes:

- quitting smoking and alcohol;
- the correct regime and nature of the diet;
- teeth sanitation, elimination of gum pockets;
- elimination of occupational hazards;
- treatment of microbial relatives.

Prophylaxis is carried out once a year in UBI, even if clinical remission is preserved; with YABDPK-1 every 2 years.

Indications for referring ulcer patients to a gastroenterologist:

- Combination of ulcer with other diseases of the digestive system.
- Determination of antibiotic resistance and antibiotic susceptibility of H. pylori strain.
- Selection of an antisecretory drug by pH-metry.
- Condition after surgical interventions with decompensated course.
- Classes at the gastronomic school.

- Determination of indications for endoscopic treatment.

CHRONIC HEPATITIS

Chronic hepatitis- long-term inflammatory liver damage, which can turn into a more severe disease - cirrhosis, remain unchanged or regress under the influence of treatment or spontaneously. The main criteria for classifying the disease as chronic hepatitis is the persistence of diffuse liver inflammation for more than 6 months.

In 1994, at the X Congress of Gastroenterologists, held in Los Angeles, a new classification of chronic liver diseases was adopted. It is based on four main criteria of the disease: etiology, pathogenesis, degree of activity and stage of the disease.

According to the etiological and pathogenetic criterion, there are:

- chronic viral hepatitis
- chronic hepatitis B
- chronic hepatitis D
- chronic hepatitis C
- chronic hepatitis of unknown viral etiology
- autoimmune hepatitis 1,2 and 3 types;
- primary biliary cirrhosis
- primary sclerosing cholangitis
- chronic drug hepatitis
- chronic cryptogenic hepatitis (i.e. chronic liver disease with morphological changes characteristic of chronic hepatitis, excluding viral, autoimmune and drug etiology).

This classification did not include such chronic diseases as alcoholic chronic hepatitis, hepatitis (liver alpha-antitrypsin deficiency, Wilson-Konovalov disease), mixed hepatitis.

The degree of activity of chronic hepatitis.

The concept of "degree of activity of the process" includes a complex of clinical data (jaundice, subfebrile condition, etc.), the level of activity of serum transaminases and the severity of the inflammatory process in the liver, which is primarily established based on the results of morphological examination of liver biopsy. The degree of process activity can be associated with the concept of the severity of the process. In the previous classification of chronic hepatitis (adopted at the congress of hepatologists in Acapulco, 1974), two morphological forms of chronic hepatitis were distinguished: chronic persistent hepatitis - CPH and chronic active hepatitis - CAH.

Chronic hepatitis stagereflects the time course of the process and is characterized by the degree of liver fibrosis. At the same time, portal, periportal (formation of port-central and port-portal septa) and perihepatocellular fibrosis are distinguished. The final stage in the development of fibrosis is liver cirrhosis. To quantify the severity of fibrosis, scoring systems have been developed.

In the course of the disease, periods of remission alternate with periods of exacerbation, which determines the variety of clinical manifestations. So, if during the period of remission in patients with chronic hepatitis of low activity the clinical condition is usually satisfactory, then during periods of exacerbation (relapses) they have weakness,

pain in the liver, dyspeptic disorders, joint pain, itching of the skin. In chronic hepatitis with high activity, the clinical manifestations of the disease, both during periods of remission and relapse, are more pronounced. An active process can lead to complete disability. The disease proceeds with severe jaundice, fever, the appearance of hepatic signs - "spider veins", palmar erythema.

The outcomes of chronic hepatitis can be - recovery, in particular - spontaneous, long-term remission, cirrhosis of the liver and the development of primary liver cancer.

Among liver diseases, viral etiology lesions occupy the main place. Currently, hepatitis viruses A, B, C, D, E, F, G have been identified, the most numerous of which are the first 4. Depending on the nucleic acid included in the structure of the virus, RNA-containing (A, C, D, E) and DNA-containing (B) viruses. Various types of acute and chronic viral hepatitis do not have pathognomonic characteristics of the clinical course and histological picture of liver damage. Consequently, a complete diagnosis of viral hepatitis can be made only on the basis of specific laboratory tests that identify individual components of the virus or antibodies to them. Modern diagnostic kits include immunological tests (ELISA) and hybridization tests (PCR). PCR is a very sensitive technique, very informative for confirming the diagnosis in case of questionable data of immunological tests,

Detection of specific viral markers in blood serum allows: to obtain 100% confirmation of diagnoses of viral hepatitis A, B, E; to identify infection with hepatitis delta (D) and C; diagnose anicteric and subclinical forms of hepatitis; diagnose mixed infections (a combination of diseases with viral hepatitis A and B), coinfection (with simultaneous infection with viral hepatitis B and D), superinfection (layering of viral hepatitis D on a chronic infection caused by the hepatitis B virus);

Objective, timely, accurate diagnosis of viral hepatitis of various etiologies provides:

- development of clear therapeutic tactics and specific preventive measures;
- etiological decoding of chronic liver damage;
- identification of virus carriers among donors and risk groups.

Viral hepatitis B

Viral hepatitis B is a widespread human infection caused by the hepatitis B virus. Hepatitis B virus (HBV) is a member of the hepatnavirus family. The HBV virion is a specific particle with d = 42 HM, consisting of a nucleoid core, inside which there is a double-stranded DNA, a terminal protein, and an enzyme DNA polymerase.

Reproduction of HBV occurs in the hepatocytes of the liver. The source of HBV infection is a patient at all stages of an acute infectious process, carriers of HBsAg. Hepatitis B is ubiquitous. Children under 1 year old and the adult population are most often involved in the epidemiological process. The main routes of transmission of the virus are parenteral, sexual, transplacental. A baby can become infected while passing through the birth canal or breastfeeding. All secretions that are formed in the patient are potentially infectious. The virus infects not only hepatocytes, but also peripheral blood lymphocytes and, according to some scientists, other organs and tissues. In acute HBV, the virus appears in the blood 2-8 weeks before the appearance of the clinic and an increase in transaminase (ALaT) activity.

Viral hepatitis C

Hepatitis C is an infection caused by the hepatitis C virus. In clinically severe cases, it is characterized by symptoms of acute liver damage, most often proceeding with moderate intoxication. The virion of the hepatitis C virus from the flavivirus family has a

diameter of 32 NM, is inactivated at 60 C within 30 minutes, and at 100 ° C in 2 minutes. A feature of the hepatitis C virus is the heterogeneity of its genome. Sources of the virus are patients with all forms of acute and chronic hepatitis C, as well as virus carriers. Routes of transmission: parenteral (with blood transfusions, medical invasive manipulations), vertical (transmission of the virus from the mother to the fetus), sexual route. The incubation period of viral hepatitis C is from 2 to 26 weeks.

Clinically, viral hepatitis C is much easier than B, often in an anicteric form. It is believed that for one case of the disease proceeding with jaundice, there are 2 cases of anicteric hepatitis. Often, the acute clinical form is not recognized due to the asymptomatic course. The icteric period is often acute without pronounced symptoms of intoxication. The mortality rate for hepatitis C is 1-2%.

This disease is especially unfavorable because of its tendency to chronicity in 30-50% of cases. A characteristic sign of HCV is the frequent development of chronic hepatitis - in 50 -70%, or cirrhosis of the liver in 30% of those who have been ill. The question of the duration of the persistence of the hepatitis C virus in the human body remains open. Currently, hepatitis C virus infection is considered a factor contributing to the development of primary hepatocellular carcinoma.

Antibodies to the hepatitis C virus begin to be produced by the body (and detected by enzyme-linked immunosorbent assay systems) 2 to 6 months after infection. An earlier diagnosis is provided by PCR with detection of viral RNA.

Viral hepatitis D

The hepatitis D or delta virus is represented by spherical particles 35-37 NM in diameter, the outer shell of which is represented by HBsAg. The hepatitis D virus is a virus because it cannot reproduce on its own. The hepatitis B virus acts as a helper virus, providing fixation and penetration of the D virus into liver cells. Delta infection exists in 2 forms:

- acute infection with simultaneous infection with hepatitis B virus and deltavirus - coinfection;
- acute infection with hepatitis D virus infection of HBsAg carriers - superinfection.

Transmission of IOP occurs with blood, sexually, vertical transmission from mother to newborn is much less common. HDV infection can cause the development of fatal fulminant or acute hepatitis with a successful course that ends in recovery. Chronic hepatitis develops in 1-3% of patients who underwent delta-hepatitis in the form of coinfection, and in 70-80% - superinfection. Detection of delta infection is a poor prognostic sign indicating an outcome in liver cirrhosis.

Clinical manifestations, features of the course of chronic hepatitis

Infection with the hepatitis B virus in a susceptible organism leads to the development of an acute or chronic infection. Acute hepatitis can occur in an icteric, anicteric or subclinical form, which in most cases can be diagnosed only by determining serological markers of hepatitis B virus infection.

The most frequent variant of the course is an acute cyclic form, in which four periods are distinguished: incubation, prodromal (preicteric), icteric (the height of the disease) and convalescence.

The incubation period ranges from 30 days to 6 months (most often 60-120 days). It depends on many reasons, the main of which are: the concentration of the virus in the infectious material, the individual susceptibility of the organism, concomitant diseases,

etc.

The prodromal period can last from 1-5 days to 1 month. Typical symptoms in this case are: decreased appetite, weakness, nausea, constipation, alternating with diarrhea, a feeling of heaviness, and sometimes pain in the right hypochondrium, musculoskeletal pain (in 50% of patients), and in some patients, urticarial rashes and itching of the skin. In the final days of the prodromal period, the liver and spleen are enlarged. The concentration of urobilinogen increases in urine. In the blood serum, the activity of transaminases is increased. There is a mild leukopenia, without changes in the leukocyte formula.

The characteristic biochemical indicators of the icteric period of acute hepatitis B are: hyperbilirubinemia; a mandatory increase in the activity of serum transaminases (their level does not depend on the severity of the disease) and an increased concentration of bilirubin. Thymol test values are usually normal. During the icteric period, leukopenia, lymphocytosis, and a decrease in ESR values up to 2-4 mm / hour are recorded in the peripheral blood.

In cases where the disease lasts more than 6 months, it is regarded as chronic hepatitis B. Its development can continue for many years. In 15-20% of cases, patients have a gradual (5-20 years) progression to cirrhosis, and in some of them to primary hepatocellular carcinoma. The reasons leading to the development of a chronic process have not been finally established.

It is believed that, first of all, this may be due to the presence of disorders in the cellular link of immunity in the patient and to the low production of endogenously synthesized interferon.

The spectrum of manifestations of chronic hepatitis B is wide, from mild (chronic lobular and chronic persistent hepatitis) to severe chronic hepatitis (chronic active hepatitis with bridging necrosis). In this regard, the clinical symptoms are extremely diverse: weakness, fatigue, malaise, arthralgia, myalgia, urticarial rashes, nausea, anorexia, weight loss in severe cases, sometimes low-grade fever, etc. At the later stages of the development of chronic hepatitis during the formation of liver cirrhosis is recorded jaundice, dark urine, spider veins, enlarged liver and spleen.

In most cases, at the initial stages of development, chronic hepatitis B occurs without jaundice. Due to the absence or minimal manifestation of clinical symptoms, the patient does not pay attention to his disease for a long time (sometimes for several years). In the case of an uncomplicated course of the disease, the indicators of the activity of serum transaminases are within a 4-fold increase in comparison with the upper limit of normal values. With the progression of chronic hepatitis B, an increase in the activity of transaminases, an increase in the concentration of gamma globulins in blood serum, alkaline phosphatase, gammaglutamyl transpeptidase, a decrease in the concentration of serum albumin, and a decrease in the prothrombin index are recorded. In some cases, the value of the activity of Al AT and AsAT may exceed 1000 IU / L.

Currently, when characterizing chronic hepatitis B, both clinical and morphological indicators are taken into account, using such terms as: "mild", "moderate", "severe". In addition, two variants of the course of chronic hepatitis B are distinguished - with high and low replicative activity of the hepatitis B virus. This activity is judged by the detection of serological markers of active viral replication: HBV DNA and HBeAg. During chronic hepatitis B, there are three phases (periods):

- the phase of immune tolerance (replicative phase), in which HBV DNA replicates with the synthesis of viral antigens: HBeAg, HBcAg and HBsAg. Morphologically, this phase is characterized by indicators characteristic of chronic persistent hepatitis;
- the phase of immune cytolysis (seroconversion), in which lysis of hepatocytes

occurs, on the membrane of which there is HBeAg. During this period, seroconversion from HBeAg to anti-HBe is recorded;

- integration phase. In some patients, lysis of all infected hepatocytes does not occur. HBV DNA is integrated into the hepatocyte genome. The level of viral replication decreases, but the synthesis of HBsAg continues.

The course of chronic hepatitis B is characterized by periods of exacerbation and remission, which can be of varying duration.

Hepatitis D, (Hepatitis D)- Delta hepatitis - an infection caused by the hepatitis D virus (BHD), characterized by symptoms of acute liver damage and intoxication, in most cases more severe than other viral hepatitis. A prerequisite for the manifestation of the pathological effect of IOP is the presence of a replicating hepatitis B virus.

Pathomorphologically, hepatitis D has no specific signs and is characterized by general manifestations of inflammation and necrosis. At the same time, necrotic changes in hepatocytes are more pronounced in the absence of a clearly expressed inflammatory reaction. With coinfection, changes characteristic of acute hepatitis B are recorded.In cases of acute superinfection, there are signs of acute inflammation and a chronic process caused by a previous infection of hepatitis B.

Acute delta hepatitis may result in recovery or chronic hepatitis. As with other viral hepatitis, diagnostic markers of infection are of great importance for the diagnosis and control of the course of the disease. These include: delta antigen, IgG and IgM antibodies to it, HDV RNA).

Chronic hepatitis develops in 1-3% of patients who underwent delta-hepatitis in the form of coinfection, and in 70-80% - superinfection.

Chronic delta hepatitis does not have clinical symptoms that are strictly characteristic only for this disease. As in other chronic hepatitis, polymorphism of clinical signs is recorded: jaundice, weakness, secondary hepatic signs - large "stars" on the face, back, upper shoulder girdle, palmar erythema, enlargement of the liver and spleen with a thickened consistency. Almost all patients have hemorrhagic syndrome - bleeding of the gums, frequent nosebleeds, a tendency to hematomas (bruises). Hemorrhagic syndrome is also associated with damage to hepatocytes, in which there is a violation of the synthesis of components of the blood coagulation system (prothrombin, factor VII, heparin, etc.).

In chronic delta-hepatitis (especially during periods of exacerbation), a change in immunological parameters occurs: a decrease in the number of T-lymphocytes with a decrease in their functional activity, a decrease in the interferon-producing ability of lymphocytes.

It seems extremely important that more severe liver damage is more often recorded in patients with chronic delta-hepatitis, who have no (or decreased) rates of active HBV replication.

Chronic delta-hepatitis can have three variants of the course: with slow (10 years or more) progression into chronic active hepatitis; rapid progression (1-2 years) and a relatively stable course of the process at the CAH level (up to 10 years). The main outcome of chronic delta infection is liver cirrhosis. Severe liver damage is the cause of high mortality in this infection.

Hepatitis C (Hepatitis C)- an infection caused by the hepatitis C virus; in clinically severe cases, it is characterized by symptoms of acute liver damage, which most often proceeds with moderate intoxication and in most cases ends with the development of chronic hepatitis with a possible transition to cirrhosis and primary liver cancer. Previously,

hepatitis C was referred to as "Hepatitis nA, ni-B".

One of the main characteristics of hepatitis C is frequent chronicity. It is believed that 60-70% of acute hepatitis C ends in the development of chronic hepatitis. The transition from acute to chronic hepatitis C occurs gradually. For several years, the activity of the pathological process and liver fibrosis has been increasing. Indicators of activity of serum transaminases within normal limits or slightly increased. Indicators of synthetic liver function (the amount of total protein and albumin) are within normal limits, up to the development of liver cirrhosis. The factors determining the development of chronic hepatitis can be attributed to the age of the patient with acute hepatitis C.

Chronic hepatitis C is characterized by the presence of lymphoid infiltrates in the liver; the existence of patients with HCV viremia, but with normal serum transaminase levels recorded for a long time. In addition, it has been suggested that increased levels of iron deposition in liver cells may play a role in liver damage.

Pathomorphologically, hepatitis C is characterized by general manifestations of inflammation and necrosis. At the same time, unlike hepatitis B or A, as well as hepatitis of non-viral etiology, hepatitis C is characterized by such histological signs as: the presence of dense lymphocytic aggregates and follicles in the portal tracts; intravascular sinusoidal infiltrates of lymphocytes or hyperplastic Kupffer cells in the absence of pronounced necrosis of hepatocytes in the immediate environment; altered epithelium of the bile ducts; fatty degeneration. At the same time, it should be borne in mind that the spectrum of damage to liver cells in hepatitis C can be extremely wide.

Most patients with chronic hepatitis C have an asymptomatic course for a long period of time. In 55-60% of cases, hepatic manifestations of the disease are recorded. These include: a slight increase in the liver, an increase in the activity of serum transaminases (2-3 times), alternating with periods of their normalization. During the period of clinically pronounced symptoms of the disease, the patient notes fatigue, lethargy, malaise, decreased ability to work, poor sleep, a feeling of heaviness in the right hypochondrium. In 40-45% of cases, extrahepatic manifestations of the disease are recorded. Other extrahepatic manifestations of chronic HS infection include: endocrine (hyperthyroidism, hypothyroidism, Hashimoto's thyroiditis); hematological (idiopathic thrombocytopenia, aplastic anemia, etc.); damage to the salivary glands and eyes (lymphocytic sialoadenitis, corneal ulcers, uveitis); cutaneous (late cutaneous porphyria, lichen planus, erythema nodosum, etc.); neuromuscular and articular (myopathic syndrome, Guillain-Barré syndrome, etc.); renal (glomerulonephritis); autoimmune (periarteritis nodosa). In most cases, extrahepatic manifestations are recorded in patients prone to autoimmune reactions. It is now also accepted that infection with the hepatitis C virus is the cause of the development of hepatocellular carcinoma. In most cases, extrahepatic manifestations are recorded in patients prone to autoimmune reactions. It is now also accepted that infection with the hepatitis C virus is the cause of the development of hepatocellular carcinoma. In most cases, extrahepatic manifestations are recorded in patients prone to autoimmune reactions. It is now also accepted that infection with the hepatitis C virus is the cause of the development of hepatocellular carcinoma.

Autoimmune hepatitis- inflammation of the liver tissue due to autoimmune disorders. Until now, there is no single point of view on the etiology of this disease. Synonyms for autoimmune hepatitis: immunoaggressive, lupoid hepatitis. Currently, the following criteria are used to identify autoimmune hepatitis: cryptogenic (unknown) nature of hepatitis; determination of autoantibodies in blood serum; violent hypergammaglobulinemia; the effectiveness of corticosteroid therapy, as a result of which

there is a rapid decrease in the level of activity of serum transaminases and the concentration of bilirubin.

Depending on the detection of certain autoantibodies, there are three main types of autoimmune hepatitis:

Type 1 - in the presence of antinuclear antibodies, antibodies against smooth muscle fibers and actin;

Type 2 - in the presence of antibodies to microsomes of liver and kidney cells;

Type 3 - in the presence of antibodies to soluble hepatic antigen.

In the structure of autoimmune hepatitis, 85% of cases belong to type 1 of the disease, which is eight times more often recorded in women than in men. In patients with type 2 autoimmune hepatitis, an increased frequency of anti-HCV detection is recorded, which, according to various authors, ranges from 48 to 100%. Depending on the presence of anti-HCV, autoimmune hepatitis is divided into two subtypes - 2a (without anti-HCV and HCV RNA) and 2b (with the presence of anti-HCV and HCV RNA). The frequent detection of anti-HCV made it possible to suggest a role for the hepatitis C virus in the development of this disease.

Clinically, autoimmune hepatitis is characterized by a wide range of manifestations: from asymptomatic to severe and fulminant hepatitis. In autoimmune hepatitis, various extrahepatic manifestations can be reported.

An autoimmune component may also be present in chronic hepatitis caused by hepatitis B, C and D viruses. This is evidenced by the detection of autoantibodies in the blood serum of these patients.

Currently, it is assumed that the viruses responsible for the development of hepatitis may play a trigger (i.e., triggering) role in the development of autoimmune hepatitis.

Currently, the diagnosis of autoimmune or viral hepatitis is of fundamental importance, since it determines the tactics of drug therapy. For the treatment of autoimmune hepatitis, immunosuppressive drugs are used: prednisolone and azathioprine, and for the treatment of viral chronic hepatitis - interferon drugs.

Drug induced hepatitis- hepatitis due to the toxic effect of the drug. Almost any drug can be the etiological agent of medicinal hepatitis. Most often, drug hepatitis is associated with the intake of psychopharmacological agents (derivatives of phenathiosin, etc.), azathioprine, contraceptive drugs, anabolic steroids, tranquilizers.

Hepatitis can occur in both acute and chronic form. Clinical and morphological manifestations of medicinal hepatitis are very diverse.

The characteristic clinical signs of drug-induced hepatitis are hepatomegaly and cholestasis. The duration of the icteric period is extremely variable - from 1-4 weeks to several months.

Chronic hepatitis treatment

An obligatory component of modern therapy for patients with chronic diffuse liver diseases is nutritional therapy. The treatment table N5 includes proteins - 90-100 g, fats - 80 g and carbohydrates - 350-400 g (in terms of 1 kg of body weight, about 1.5-1.2-5.5 g / day). Food must contain all essential vitamins and minerals.

Chronization of viral infection is observed in patients with impaired cellular immunity and low production of endogenous interferon.

In chronic viral hepatitis B and C in cases of high replicative activity, treatment with interferon is indicated. The problem of interferon therapy has been going on for more than 20 years and has attracted the widest attention in recent years, and although a huge

literature has now accumulated that summarizes the experience of many countries, it is far from its final solution, and assessments of its effectiveness are very contradictory. The goals of antiviral therapy for chronic viral hepatitis are the elimination or cessation of viral replication, arrest or decrease in the degree of inflammation activity, prevention of the progression of chronic hepatitis with the development of long-term consequences, including cirrhosis and hepatocellular carcinoma.

Prednisolone is prescribed orally for 7-8 weeks: the first 2 weeks at 60 mg, 3-4 weeks at 40 mg and the last two at 20 mg daily. According to another scheme, treatment with prednisolone is continued for 4 weeks: the first 3 weeks, 30 mg; 4-week -15mg / day.

Essentiale is a drug of membrane stabilizing and lipotropic action, used intravenously in 2-4 ampoules of 5 ml daily for 4-6 weeks or 1-2 capsules 3 times a day for up to 12 weeks.

Flavonoids are preparations from the fruits of Silybium marianum, produced in the form of pills and tablets under different names: silymarin, silibinin, silibor, carsil, legalon.

Hepabene is a mixture of milk thistle and fume extract. Assign inside 1-2 capsules 3 times a day for 4-12 weeks.

Hepatofalk - is a combination of 3 medicinal herbs - thistle, celandine and Japanese turmeric. Assign 1-2 capsules 3 times / day for 4-12 weeks.

Ursofalk - the active ingredient is ursodeoxycholic acid. Dosage: 10 mg / kg body weight per day (one capsule before bedtime).

Tykveol is a pumpkin seed lipid complex. Assign 1 teaspoon 3-4 times a day on an empty stomach for 3-4 weeks.

Thioctacid - thioctic acid, is prescribed at 600 mg (one tablet) 30 minutes before meals as maintenance therapy after 2-4 weeks of intravenous administration of 1 ampoule of thioctacide.

Along with drug therapy, which has recently been using phytopreparations, methods of efferent treatment of patients with chronic diffuse liver diseases are increasingly being used: hemosorption, lymphosorption, plasmapheresis, blood ultraviolet irradiation, magnetotherapy, laser therapy, ozone therapy, etc. Non-drug therapy of hepatitis and liver cirrhosis is effective and very promising. These methods deserve wider implementation in clinical practice.

CIRRHOSIS OF THE LIVER

Cirrhosis of the liver - a chronic progressive disease characterized by damage to both the parenchyma and the stroma of the organ with dystrophy of the liver cells, nodular regeneration of the liver tissue, the development of connective tissue, diffuse rearrangement of the lobular structure and vascular system of the liver.

Etiology

Liver cirrhosis can be the result of a huge number of factors that cause damage to hepatocytes and their necrosis, and this process can be either gradually progressive or recurrent.

In our country, the leading role in the development of this disease is played by viral liver damage (especially viral hepatitis B), in the outcome of which cirrhosis of the liver is formed (according to various statistics, 17-70% of the total number of patients with liver cirrhosis).

For a long time, chronic alcoholism was considered one of the leading causes of liver cirrhosis. Recently, it has been proven that along with chronic alcohol intoxication in the development of this disease, the accompanying background of malnutrition or a

deficiency of proteins and vitamins in food is usually of great importance. At the same time, the specific effect of alcohol on many metabolic processes in the liver has been proven. In an experiment on animals, it was shown that long-term alcohol intoxication, despite good nutrition, causes changes in some enzymatic processes and dystrophic changes in hepatocytes.

Alimentary factor - mainly a deficiency of proteins and vitamins (especially B and folic acid) - is one of the common causes of liver cirrhosis in a number of countries with tropical and subtropical climates. In some cases, malnutrition is of endogenous origin, associated with a malabsorption of proteins and vitamins in the gastrointestinal tract. In countries with a tropical climate, cirrhosis often occurs against the background of chronic parasitic and helminthic lesions of the liver. Toxic cirrhosis of the liver can occur with repeated and prolonged exposure to hepatotoxic substances, with food poisoning. The group of toxic-allergic cirrhosis also includes lesions associated with hypersensitivity to various drugs, resulting in liver cell necrosis.

Biliary cirrhosis of the liver develops as a result of obstruction of the intra- and extrahepatic bile ducts and their inflammation, which leads to stagnation of bile (cholestasis). The cause of cholestasis is often chronic cholangitis, accompanied by deformation and obstruction of the intra- and extrahepatic bile ducts (the so-called primary biliary cirrhosis), as well as other reasons: compression (by a tumor) or prolonged blockage of large (extrahepatic) ducts with gallstones, helminths, etc. . (secondary biliary cirrhosis). In a number of cases, in primary biliary cirrhosis, bile secretion defects are observed due to impaired hepatocyte function.

Metabolic and endocrine factors can also cause the development of liver cirrhosis (thyrotoxicosis, diabetes mellitus, etc.).

In some cases, the etiology of liver cirrhosis is mixed; the disease occurs as a result of the simultaneous effect of several factors on the body. The etiological factor does not in all cases determine the pathways of development of liver cirrhosis. One and the same damaging agent can lead to the formation of different morphological variants of cirrhosis, and different etiological factors - to similar morphological changes.

Finally, relatively often (in 20-30% of patients), the cause of liver cirrhosis remains unclear. However, in this group of patients, viral hepatitis that has been transferred in the past, proceeding in an erased, anicteric form, is possible.

For a long time, cirrhosis of the liver was attributed to its lesions arising from a violation of the outflow of blood through the hepatic veins (with cardiac venous stasis, thrombophlebitis of the hepatic veins, etc.). In these cases, the development of connective tissue in the liver and an increase in its size are also observed. However, there is usually no nodular regeneration of the hepatic parenchyma, therefore such lesions of this organ are referred to as "pseudocirrhosis" or "liver fibrosis".

Pathogenesis

The origin of the disease in many cases is associated with a long-term direct effect of the etiological factor (virus, intoxication, etc.) on the liver, circulatory disorders in it. The development of connective tissue in the form of cords and scars, which change the normal architectonics of the liver, leads to compression of its vessels, disruption of the normal blood supply to the liver cells; hypoxia contributes to further disturbances in the normal course of redox enzymatic reactions in the liver tissue, enhances dystrophic changes and, contributing to the progression of the process, creates a vicious circle. Products of necrobiotic decay of hepatocytes contribute to regenerative processes, as well as the development of an inflammatory reaction. In the progression of cirrhosis of viral etiology,

apparently,

Simultaneously with the compression of the hepatic vessels by the nodules of the regenerating hepatic parenchyma and connective tissue cords, the number of anastomoses between the branching of the portal and hepatic veins, as well as the hepatic artery, facilitating local intrahepatic circulation, increases. At the same time, blood through these anastomoses bypasses the preserved hepatic parenchyma, which sharply impairs its blood supply and can lead to new ischemic necrosis, secondary collapses, i.e., to the progression of cirrhosis.

In the progression of chronic liver diseases and the development of cirrhosis, immune disorders are also important, manifested in the acquisition of antigenic properties by some modified proteins of hepatocytes and the subsequent production of autoantibodies to them. Antigen - antibody complexes, fixing on hepatocytes, cause their further damage.

The classification of liver cirrhosis is a complex problem. Previously, the classification adopted by the Pan American Congress of Gastroenterologists in 1956 was used, according to which portal, postnecrotic, biliary and mixed cirrhosis was distinguished.

In the future, the World Association for the Study of Liver Diseases (Acapulco, 1974) and WHO (1978) recommended using a classification based on etiological and morphological principles; it formed the basis of the classifications.

A. By etiology, cirrhosis is distinguished:

a) due to viral liver damage;

b) due to malnutrition;

c) due to chronic alcoholism;

d) cholestatic;

e) as an outcome of toxic or toxic-allergic hepatitis;

f) constitutional and familial cirrhosis;

g) due to chronic liver infiltration with certain substances with subsequent inflammatory reaction (hemochromatosis, Konovalov-Wilson disease);

h) cirrhosis developing against the background of chronic infections (tuberculosis, syphilis, brucellosis), and cirrhosis of other etiology, including those arising from unexplained causes (cryptogenic).

B. According to morphological and partly clinical signs, micronodular or small-nodular cirrhosis (basically corresponds to portal cirrhosis of the previous classifications), macronodular, or large-nodular cirrhosis (according to many signs corresponds to postnecrotic cirrhosis), mixed and, finally, biliary (primary and secondary) cirrhosis of the liver ...

B. According to the activity of the process, cirrhosis is distinguished:

a) active, progressive;

b) inactive.

D. According to the degree of functional disorders, cirrhosis is distinguished:

a) compensated;

b) decompensated.

Clinical picture

The clinic depends on the type of cirrhosis, the stage of the disease (compensated or decompensated) and the degree of activity of the pathological process in the liver. The main clinical signs of liver cirrhosis, which make it possible to distinguish it from hepatitis and other lesions of this organ, are:

1. The presence of an enlarged dense liver and spleen (in advanced cases, the size of the liver can be reduced);

2. Ascites and other signs of portal hypertension

3. The so-called hepatic stigmas, especially cutaneous hepatic "asterisks" (telangiectasias).

With various variants of cirrhosis, pain occurs in the liver, in the epigastric region or throughout the abdomen, have a dull aching character, intensify after eating, especially fatty, drinking and physical work. The cause of pain is usually an enlargement of the liver and stretching of the capsule, the appearance of foci of necrosis close to the capsule, and reactive involvement in the process of closely located areas of the hepatic capsule.

With cirrhosis of the liver, dyspeptic phenomena are common in the form of a decrease in appetite to complete anorexia (more often with alcoholic cirrhosis), heaviness in the epigastric region after eating, nausea, flatulence and upset stools (especially diarrhea after eating a fatty meal), rarely - severe nausea and vomiting, which mainly due to impaired bile secretion and concomitant biliary dyskinesia or alcoholic gastroenteritis. Severe flatulence is sometimes accompanied by distal abdominal pain, usually short-term attacks. Frequent complaints of patients with liver cirrhosis are decreased ability to work, general weakness, fatigue and insomnia.

Portal hypertension to a greater or lesser extent can be observed in various forms of liver cirrhosis, but it is especially characteristic of portal cirrhosis. The emergence of this syndrome is due to organic disturbance of intrahepatic circulation as a result of obstruction of venous outflow by nodes - regenerates and the formation of connective tissue septa with desolation of most of the sinusoids. As a result of these reasons, an obstacle is created for the outflow of blood from the liver, the portal pressure rises significantly - up to 400 - 600 mm of water. (normally does not exceed 120-150 mm of water. ST.). For a long time, violations of the portal circulation can be compensated by the development of anastomoses

Hemorrhagic syndrome occurs in about half of patients with liver cirrhosis. Massive bleeding from the dilated veins of the esophagus and stomach, as well as hemorrhoids, are caused by increased pressure in these veins, thinning of their walls or injury to them. They are characteristic of portal cirrhosis. Repeated nosebleeds can be one of the initial signs of liver cirrhosis. Repeated nosebleeds, uterine bleeding, skin hemorrhages are caused by blood clotting disorders as a result of impaired liver involvement in the production of certain clotting factors. They appear with severe decompensation of cirrhosis. Recently, attention has been paid to the hemodynamic disorders characteristic of this disease. With cirrhosis of the liver, high cardiac output and increased pulse pressure are noted.

Laboratory blood tests usually reveal anemia, leukopenia, thrombocytopenia, and increased ESR. Especially severe hypochromic anemias are observed after bleeding. Anemia with moderate macrocytosis may also be due to hypersplenism. In rare cases, megaloblastic anemia develops due to vitamin B12 deficiency.

A manifestation of liver dysfunction in cirrhosis is a decrease in the content of prothrombin and fibrinogen in the blood serum, the synthesis of which is carried out by the hepatic cells; usually, the antithrombin activity of plasma increases, its general coagulating activity decreases. The change in these indicators of the coagulation and anticoagulation systems of the blood reflects the tendency to hemorrhagic diathesis characteristic of liver cirrhosis.

In diagnostically difficult cases, they resort to laparoscopy and percutaneous liver biopsy. These instrumental methods make it possible to detect the characteristic morphological signs of each of the variants of liver cirrhosis. Scanning the liver allows you

to determine its size, the state of the liver's absorption function, as well as to determine the concomitant enlargement of the spleen. For the diagnosis of cirrhosis, echography is widely used, which also allows you to determine the size of the liver and spleen, their structure.

X-ray examination method reveals the expansion of the veins of the esophagus. In thin people with flatulence, sometimes during fluoroscopy, a shadow of an enlarged liver and spleen can be seen.

Although with various morphological variants of cirrhosis, all of the listed symptoms may be more or less pronounced, however, in "pure" cases, a predominance of certain of them is observed. So, with micronodular portal cirrhosis of the liver, the clinical picture of the disease is more dominated by symptoms of portal hypertension, signs of functional liver failure develop only in the very late period of the disease. For macronudular (postnecrotic) cirrhosis of the liver, relatively early arising signs of liver failure are more characteristic. The weakness is most pronounced, the phenomena of hemorrhagic diathesis are often observed, significant changes are determined by the biochemical study of blood serum (hypoprothrombinemia, hypofibrinogenemia, etc.). The liver is usually not dramatically enlarged or even reduced in size. For biliary cirrhosis of the liver, the most typical symptom with a satisfactory general condition of the patient is chronic jaundice with severe pruritus, often with high body temperature, sometimes accompanied by chills. Increased blood levels of alkaline phosphatase, cholesterol. Unlike other variants of liver cirrhosis, significant splenomegaly, portal hypertension syndrome, and spider veins are less common. All these changes, if they develop, then only in the late stage of the disease. Increased blood levels of alkaline phosphatase, cholesterol. Unlike other variants of liver cirrhosis, significant splenomegaly, portal hypertension syndrome, and spider veins are less common. All these changes, if they develop, then only in the late stage of the disease. Increased blood levels of alkaline phosphatase, cholesterol. Unlike other variants of liver cirrhosis, significant splenomegaly, portal hypertension syndrome, and spider veins are less common. All these changes, if they develop, then only in the late stage of the disease.

Flow

Liver cirrhosis usually tends to progress. However, in some cases, the progression of the disease occurs quickly and over the course of several years leads the patient to death - an active progressive process. It is often characterized by alternating periods of exacerbation (with a greater severity of morphological and biochemical changes) and periods of remission, when the patient's well-being and many clinical and laboratory parameters improve significantly. In other cases, a delayed course of the disease (tens of years) with slightly pronounced signs of activity is noted - inactive cirrhosis. Remissions can be very long, years, periods of exacerbation of the disease are not clearly differentiated or occur rarely (after significant disturbances in the diet, against the background of severe infectious diseases, etc.).

There are also decompensated and compensated cirrhosis. With compensated cirrhosis of the liver, complaints may be absent or it may proceed with mild symptoms and be identified by random examination on the basis.

Enlargement of the liver and spleen, the presence of liver "signs". Changes in laboratory parameters are also insignificant: hypergammaglobulinemia, a moderate decrease in the absorption and excretory functions of the liver, and an increase in ESR are noted.

Decompensation of cirrhosis is characterized by a sharp decrease in working

capacity, general weakness, insomnia, increased dyspeptic symptoms, weight loss, moderate fever, and hepatic breath odor. Jaundice appears or intensifies, the skin can be not only icteric, but also pigmented due to increased deposition of melanin. Hemorrhages in the skin and nosebleeds occur spontaneously. There is a tendency towards a decrease in blood pressure. Ascites develops rapidly, preceded by severe flatulence. The liver is more often or slightly enlarged, with a dense margin, or sharply wrinkled and not palpable. The spleen is enlarged. Laboratory data indicate a decrease in liver function: the serum level of bilirubin increases, the level of albumin decreases, the amount of globulins increases significantly, the activity of aminotransferases increases, the cholesterol content decreases, the prothrombin time is slowed down. In the urine, bilirubin and an increased amount of urobilin are determined.

The most frequent complications of liver cirrhosis are profuse bleeding from varicose veins of the cardiac segment of the esophagus and stomach, hemorrhoidal bleeding (with cirrhosis occurring with portal hypertension). Gastrointestinal bleeding in the form of bloody vomiting and melena occurs as a result of rupture of varicose veins in the lower third of the esophagus and the cardiac stomach. The immediate cause of variceal bleeding is physical exertion or local damage to the mucous membrane (eg, rough food). Trophic changes in the walls of the dilated vessels and the mucous membrane of the esophagus, high pressure in the veins, as well as reflux esophagitis, accompanying varicose veins of the esophagus, predispose to bleeding.

Perhaps the development of liver cancer (cancer-cirrhosis), the incidence of which, according to some sources, is close to 20%, as well as stomach ulcers, which often accompany liver cirrhosis.

The terminal period of the disease, regardless of the forms of cirrhosis, is characterized by the progression of signs of functional failure of hepatic cells with an outcome in hepatic coma. Esophageal-gastric bleeding and hepatic coma are the two most common immediate causes of death in patients with cirrhosis.

Diagnosis and differential diagnosis

The diagnosis is made on the basis of the characteristic clinical picture of the disease. It is confirmed primarily by a puncture biopsy of the liver, data from echography, scanning, computed tomography, angiography and other research methods. Liver cirrhosis is distinguished from chronic hepatitis, liver dystrophy, focal lesions in chronic infections, primary or secondary (metastatic) tumor lesions, secondary liver damage in Chiari syndrome, helminthic liver damage (primarily from liver echinococcosis), congestive liver, liver fibrosis , an aleukemic form of leukemia.

With fatty degeneration (fatty hepatosis), the liver is usually enlarged, but its edge is not as sharp as with cirrhosis. The spleen is usually not enlarged. With tumor damage to the liver, a relatively rapid increase in symptoms is noted (several months - 1-1.5 years), jaundice acquires features of predominantly mechanical, the liver gradually increases, often bumpy, with an uneven edge, the spleen is not enlarged. In cases where liver cancer occurs against the background of cirrhosis (cancer-cirrhosis), the diagnosis becomes more difficult. Laparoscopy and puncture biopsy, scanning, echography, computed tomography are of decisive importance in differential diagnosis.

Treatment

Treatment of patients with various forms of liver cirrhosis in the stage of compensation mainly consists in preventing further liver damage, high-calorie, nutritious dietary food with sufficient protein and vitamins in food, establishing a clear 4-5 meals a

day. Alcoholic drinks are prohibited. It is also necessary to pay attention to the correct organization of the working regime.

During the period of decompensation, treatment is necessarily carried out in a hospital. Prescribe diet therapy, use corticosteroid drugs (15 - 20 mg / day of prednisolone or equivalent doses of triamcinolone), vitamins. Corticosteroids are contraindicated in liver cirrhosis complicated by the expansion of the veins of the esophagus and in the combination of cirrhosis with peptic ulcers of the stomach and duodenum, reflux esophagitis. With ascites, a salt-restricted diet and periodic diuretics are prescribed. In order to prevent esophageal-gastric bleeding in patients with portal hypertension, surgical treatment is widely carried out - the imposition of additional portocaval anastomoses, which help to reduce the pressure in the portal, and, consequently, in the esophageal veins.

The occurrence of acute esophageal bleeding in cirrhosis of the liver requires urgent hospitalization of the patient in a surgical hospital and emergency measures to stop bleeding (tight esophageal tamponade through an esophagoscope, the use of a special three-channel probe like Sengstaken Blackmore with filling air and compressing the veins with a balloon, hemostatic therapy).

In case of inactive cirrhosis with signs of liver failure, liver drugs are prescribed (sirepar, 2-3 ml intramuscularly or intravenously 1 time per day, antianemin, etc.). With active cirrhosis, these drugs are not prescribed, since they can increase sensitization to the liver tissue and contribute to an even greater activation of the process. Long-term use of Essentiale gives a good effect (1-2 capsules 3 times a day). In severe hepatic impairment, treatment is carried out as in hepatic coma.

In primary biliary cirrhosis, a full-fledged diet rich in vitamins, especially vitamins A and K, is used. Lipoic acid preparations give some effect; cholestyramine (a polymer that binds bile acids in the intestine and prevents their absorption) is used to reduce painful itching.

With secondary biliary cirrhosis, surgical treatment is undertaken in order to eliminate blockage or compression of the common bile duct, to establish the outflow of bile and the disappearance of jaundice.

CHRONIC PANCREATITIS

Chronic pancreatitis - progressive inflammatory sclerotic disease of the pancreas, characterized by the gradual replacement of the organ parenchyma with connective tissue and the development of insufficiency of the exocrine and endocrine functions of the gland.

Prevalence

The incidence of chronic pancreatitis, according to clinical data, ranges from 0.2 to 0.6% in the general population. There are 7-10 new cases of chronic pancreatitis per 100,000 population per year. Epidemiological, clinical and pathological studies indicate that the incidence has increased in all countries in the last quarter of the 20th century. This is associated with an increase in alcoholism, an increase in the incidence of gastrointestinal diseases and diseases of the biliary tract.

Classification

According to the classification adopted in European countries, the following clinical forms of chronic pancreatitis are distinguished.

- Chronic calcifying pancreatitis is the most common form, accounting for 49

to 95% of all pancreatitis.

- Chronic obstructive pancreatitis is the second most common form of chronic pancreatitis.
- Chronic inflammatory (parenchymal) pancreatitis is rare.

According to the peculiarities of the clinical course (classification by A.A. Shelagurov), 5 forms of the disease are distinguished:

1) chronic recurrent pancreatitis, characterized by distinct periods of remission and exacerbations of the process;

2) painful form with constant pain dominating in the clinical picture of the disease;

3) pseudotumor form;

4) latent (painless) form.

With relatively early (and rapidly) progressive signs of pancreatic insufficiency, the

5) sclerosing form of chronic pancreatitis. In this form, subhepatic (mechanical) jaundice can be observed due to compression of the portion of the common bile duct passing through it by the compacted tissue of the gland.

There are three stages of the disease: 1 - initial; 2 - moderate; 3 - heavy (cachectic, terminal); 2 and especially 3 stages of the disease proceed with disturbance of the exocrine and often endocrine functions of the pancreas.

Pathogenetically, primary (the pancreas is the first and main object of damage, and then other organs are involved in the process) and secondary (resulting from damage to the biliary tract) pancreatitis are isolated.

Etiology

Alcohol- the main etiological factor, especially in men. Alcoholic pancreatitis is diagnosed in 25 - 50% of all cases. It has been proven that the consumption of alcohol in a daily dose of 80 - 120 ml of pure ethanol for 3 - 10 years leads to the development of chronic pancreatitis. The combination of regular alcohol consumption with smoking increases the risk of developing chronic pancreatitis. In 25 - 40% of patients (mainly women), the cause of chronic pancreatitis is diseases of the biliary system (dyskinesia, cholecystitis), diseases of the zone of the large duodenal papilla (papillitis, odditis, tumors).

- Diet with severely limited protein and fat (malnutrition), Chronic pancreatitis occurs when eating less than 30 grams of fat and 50 grams of protein per day. After a period of malnutrition, the pancreas becomes more vulnerable to other etiological factors, in particular alcohol.
- Hereditary metabolic disorders of certain amino acids (lysine, cysteine, arginine, ornithine, etc.) lead to the development of a special form, disease - congenital (familial) chronic pancreatitis.
- The pancreas can be affected by toxic substances (for example, solvents), drugs (azathioprine, hydrochlorothiazide, furosemide, mercaptopurine, methyldopa, estrogens, sulfonamides, tetracycline, NSAIDs).
- Hyperlipidemia.
- The development of chronic pancreatitis is possible in conditions of prolonged hypercalcemia, for example, with hyperparathyroidism or an

overdose of ergocalciferol.

- Infectious diseases (most often mumps).
- Pancreatic injuries, including operating ones.
- Hereditary predisposition. Families with chronic pancreatitis are more likely to develop it than the general population. A frequent combination of the disease with blood group 0 (1) was noted.

Pathogenesis

The main pathogenetic mechanism of the development of chronic pancreatitis is considered to be the activation of its own enzymes (trypsinogen, chymotrypsinogen, proelastase and phospholipase A), followed by damage to the gland tissue. This causes the development of edema, coagulation necrosis and, as a result, fibrosis of the pancreatic tissue. As a result of the death of acinar cells and obstruction of the intrapancreatic ducts, the flow of pancreatic enzymes into the duodenal lumen decreases, which leads to exocrine pancreatic insufficiency. As a result of the defeat of the islets of Langerhans, insufficiency of the endocrine function of the pancreas also develops. Clinically, this can manifest itself as the development of both hypoglycemic syndrome (due to glucagon deficiency) and diabetes mellitus (due to insulin deficiency).

Pathomorphology

Chronic calcifying pancreatitis is characterized by an uneven lobular lesion of the pancreas. The ducts, mostly small, are atresized or stenotic, protein precipitates or calcifications, stones are found in their lumen. The acinar tissue is atrophied, sometimes cysts and pseudocysts are found.

Chronic obstructive pancreatitis develops as a result of obstruction of the main or large ducts of the pancreas. The defeat of the pancreas occurs above the obstruction site, it is uniform and is not accompanied by the formation of stones inside the ducts.

Chronic parenchymal pancreatitis is characterized by the development of foci of inflammation in the parenchyma with a predominance of mononuclear cells and areas of fibrosis in the infiltrates, replacing the pancreatic parenchyma. With this form, there is no damage to the ducts and calcifications in the pancreas.

Clinical picture

The clinical picture of chronic pancreatitis consists of pain and dyspeptic syndromes, insufficiency of the exocrine and endocrine functions of the gland.

Pain syndrome

The localization of pain in chronic pancreatitis depends on the predominant lesion of one or another anatomical part of the pancreas.

Pain in the left hypochondrium or to the left of the navel occurs when the tail of the pancreas is affected.

When the body of the pancreas is affected, the pain is localized in the epigastric region.

When the head of the pancreas is affected, the pain is localized in the Shoffard zone.

With total damage to the organ, pains are diffuse in the form of a "belt" or "half-belt" in the upper abdomen.

Pain occurs or intensifies 40-60 minutes after eating (especially plentiful, spicy, fried, fatty). The pain increases with the supine position and decreases with the sitting position with a slight forward bend of the trunk. Possible irradiation of pain in the region of

the heart, in the left scapula, left shoulder (which imitates angina pectoris), and sometimes in the left iliac region. By the nature of the pain, it can be sudden, sharp, with a gradual intensification or constant, dull, pressing, intensifying after eating.

Dyspeptic syndromecharacterized by belching, heartburn, nausea. These signs are associated with dyskinesia of the descending duodenum and duodenostasis.

Exocrine insufficiency

Exocrine pancreatic insufficiency is characterized by a violation of the processes of intestinal digestion and absorption, the development of excessive microbial growth in the small intestine. As a result, patients develop diarrhea, steatorrhea, flatulence, abdominal pain, belching, nausea, occasional vomiting, loss of appetite, weight loss, later symptoms characteristic of polyhypovitaminosis join.

An early sign of exocrine pancreatic insufficiency is steatorrhea. Mild steatorrhea, as a rule, is not accompanied by clinical manifestations. With pronounced steatorrhea, diarrhea appears 3 to 6 times a day, the feces are mushy, fetid, with a greasy sheen. However, if the patient reduces the intake of fatty foods or uses pancreatic enzymes during treatment, then steatorrhea decreases and may even disappear. Much less often in patients with chronic pancreatitis, watery diarrhea is observed.

In a significant part of patients, weight loss is noted due to a violation of the processes of digestion and absorption in the intestine, as well as in connection with loss of appetite, a strict diet, sometimes even starvation for fear of provoking a pain attack, a deficiency of fat-soluble vitamins (vitamins A, D, E and K) occurs rarely, mainly in patients with severe and prolonged steatorrhea. Some patients with chronic pancreatitis have a vitamin deficiency B 12. However, clinical signs of vitamin B 12 deficiency are rare, since the appointment of enzyme preparations and foods rich in this vitamin, very quickly compensates for this violation.

Endocrine insufficiency

In about 1/3 of patients, disorders of carbohydrate metabolism in the form of hypoglycemic syndrome are revealed, in half of them clinical signs of diabetes mellitus are observed. The development of these disorders is based on the damage to the cells of the islet apparatus, as a result of which there is a deficiency of not only insulin, but also glucagon. This explains the features of the course of pancreatogenic diabetes mellitus: a tendency to hypoglycemia, the need for low doses of insulin, the rare development of ketoacidosis, vascular and other complications.

Depending on the form of the disease, one of the above syndromes may prevail. Constant pain syndrome is characteristic of chronic obstructive pancreatitis. Chronic inflammatory pancreatitis is characterized by slowly progressive signs of exocrine and endocrine insufficiency and the absence of pain. Chronic calcifying pancreatitis is characterized by a recurrent course with episodes of exacerbation, in the early stages resembling acute pancreatitis with severe pain syndrome.

An objective examination reveals dryness and peeling of the skin, glossitis, stomatitis (changes caused by hypovitaminosis). As a rule, patients are underweight. On the skin of the chest, abdomen, back, sometimes you can find small bright red spots of a round shape, 1 - 3 mm in size, which do not disappear when pressed (Tuzhilin symptom). It is possible to palpate the pancreas only with cystic and tumor processes. Local palpation tenderness in the pancreas is detected in half of the patients. A positive phrenicus symptom is determined.

Laboratory research methods

Laboratory research methods are used to identify the inflammatory process in the pancreas, assess its activity and determine the degree of violation of the exocrine and endocrine functions of the organ.

Identification of the inflammatory process and assessment of its activity

- In a general blood test, leukocytosis and an increase in ESR are possible
- Increased activity of pancreatic enzymes: amylase, trypsin and lipase (more specific than the definition of amylase) in serum and urine, elastase in blood and feces.
- Changes in indicators of pancreatic secretion (enzymes, hydrocarbons, juice volume) when examining the contents of the duodenum before and after stimulation with secretin, pancreozymin (hypersecretory type of secretion in the initial stages of chronic pancreatitis and hyposecretory - with the development of gland fibrosis).

Identification of exocrine pancreatic insufficiency

- Stool examination. Characterized by polyfecal matter (over 400 g / day, the results are reliable with daily weighing of feces for 3 days), steatorrhea, creatorrhea (10 or more muscle fibers in the field of view; a sign of severe pancreatic insufficiency).
- Secretin-pancreosiminovy (cerulein) test. The pancreas reacts to the introduction of pancreosimin (dose 1 U / kg) or cerulein (dose 75 ng / kg) by secreting a small volume of juice, rich in enzymes and poor in bicarbonate. After the administration of the hormone, the duodenal contents are collected for 40 minutes, after which secretin is injected (in response, iron secretes a lot of juice rich in bicarbonate and poor in enzymes) at the rate of 1 U / kg, and the contents of the duodenum are collected for another 80 minutes.
- The PABA test (benthiramine) evaluates the result of taking 0.5 g of para-aminobenzoic acid tripeptide (benthiramine). The result is considered pathological if less than 50% of bentiramine is excreted in the urine within 6 hours. Sulfonamides, meat, lingonberries, pancreatic enzymes distort the results of the study.
- The Lund Test is based on the ability of a special mixture (15 g of olive oil, 15 g of milk powder, 45 g of glucose, 15 ml of fruit syrup, up to 300 ml of distilled water) to induce endogenous production of secretin and pancreozymin - hormones produced by the duodenal mucosa. Duodenal juice is collected for 2 hours - 30 minutes before the introduction of the stimulus (basal secretion) and 90 minutes after it (stimulated secretion).

Identification of endocrine pancreatic insufficiency

The detection of endocrine pancreatic insufficiency is reduced to determining the concentration of glucose in the blood plasma and the glycemic profile (two-humped type of glycemic curve, characteristic of diabetes mellitus).

Instrumental research methods

Instrumental research methods are aimed at visualizing changes in the pancreas, the zone of the large duodenal papilla, large bile ducts, and the main pancreatic duct. Also, instrumental methods are important to exclude volumetric formations of the pancreas

(tumors, cysts).

- study of the stomach and duodenum with contrasting reveals dyskinesia, duodenostasis, changes in the position and shape of the duodenum, duodenography in conditions of hypotension (X-ray examination in conditions of artificial hypotension of the duodenum caused by the administration of anticholinergics) allows detecting an increase in the head of the pancreas.

- Ultrasound is used to identify the size, uneven contours, reduced echogenicity, heterogeneity of the structure Revealing changes in the size, configuration and density of the pancreas
- X-ray research methods. Plain X-ray of the abdominal organs allows you to detect calcifications in the tissue of the pancreas.
- CT is used with insufficient information content of previous methods.

To visualize the zone of the large duodenal papilla and large bile ducts, FEGDS is performed with an examination of the zone of the large duodenal papilla, intravenous cholegraphy, and radionuclide cholecystography.

To detect changes in the large pancreatic duct, endoscopic retrograde cholangiopancreatography is used. This study allows you to detect signs of diffuse changes in the duct system; alternation of extensions and contractions of the large pancreatic duct, tortuosity and unevenness of the walls, deformation of the lateral branches.

To exclude volumetric processes, first of all, ultrasound, CT, gland biopsy, and in some cases, angiography of the vessels of the pancreas are used.

Treatment

During the period of exacerbation, patients with chronic pancreatitis need hospitalization, constant monitoring with the control of hemodynamic and biochemical (amylase, lipase, glucose levels) indicators. Based on the pathogenesis of chronic pancreatitis, treatment should be aimed at reducing pancreatic secretion, relieving pain, carrying out enzyme replacement therapy and preventing complications.

Normalization of pancreatic secretion. Decrease in pancreatic secretion is achieved through the use of diet and remedies.

Diet

With pronounced exacerbations, hunger (zero diet) and hydrocarbonate-chloride waters are prescribed for the first 3 to 5 days, which leads to a decrease in pancreatic secretion. If necessary, they switch to parenteral nutrition (albumin, protein, glucose), which helps to reduce intoxication, pain syndrome and prevents the development of hypovolemic shock. With duodenostasis, gastric contents are aspirated, histamine H2 receptor blockers (ranitidine 50 mg 4 times a day or famotidine 20 mg 4 times a day) or pirenzepine (10 mg 4 times a day) are administered parenterally. After 3 - 5 days, the patient is transferred to oral nutrition. Eating should be frequent, small portions. Limit the intake of foods that can stimulate the secretion of the pancreas (fats, acidic foods), dairy products rich in calcium (cottage cheese, cheese). The daily diet should include 80 - 120 g of easily digestible proteins (eggs, boiled meat, fish), 80 g of fat (of which 70% are vegetable, 30% of animals), 300 - 400 g of carbohydrates (preferably in the form of polysaccharides). With good individual tolerance, raw vegetables are not excluded. Alcohol, spicy food, canned food, carbonated drinks, sour fruits and berries, sour fruit

juices are prohibited.

Medicines

Since one of the mechanisms for stimulating pancreatic secretion is an increased release of hydrochloric acid, drugs that reduce gastric secretion are used in chronic pancreatitis. Usually, non-absorbable antacids (aluminum, magnesium containing), selective M-anticholinergics (pirenzepine), histamine H2 receptor blockers (ranitidine, famotidine) are used in usual doses. Also, agents are used that suppress the activity of pancreatic enzymes and reduce their destructive effects on the pancreas and other organs and tissues. Trasisol is usually used at a dose of at least 100,000 U / day, contrikal 20,000 - 40,000 U / day, gordox 50,000 - 100,000 U / day. These drugs are used for severe exacerbations of chronic pancreatitis (pain syndrome, hyperamilasemia, hyperamylazuria), intravenous drip at a rate of 40 - 60 drops per minute after dilution in 400 ml of 5% glucose solution or isotonic sodium chloride solution for 7 - 10 days. They also use aminocaproic acid, 100 ml of a 5% solution 1-2 times a day, intravenously drip in a course of 5 - 10 days. In addition, drugs are used that reduce the pressure in the pancreatic duct system (M-anticholinergics and myotropic antispasmodics). In case of dyspeptic manifestations (heartburn, belching, nausea), drugs are used that eliminate violations of gastroduodenal motility: metoclopramide (domperidone) 10 mg 2 - 3 times a day, sulpiride 100 mg IM 3 times a day. They also use aminocaproic acid, 100 ml of a 5% solution 1-2 times a day, intravenously drip for 5 - 10 days. In addition, drugs are used that reduce the pressure in the pancreatic duct system (M-anticholinergics and myotropic antispasmodics). In case of dyspeptic manifestations (heartburn, belching, nausea), drugs are used that eliminate violations of gastroduodenal motility: metoclopramide (domperidone) 10 mg 2 - 3 times a day, sulpiride 100 mg IM 3 times a day. They also use aminocaproic acid, 100 ml of a 5% solution 1-2 times a day, intravenously drip for 5 - 10 days. In addition, drugs are used that reduce the pressure in the pancreatic duct system (M-anticholinergics and myotropic antispasmodics). In case of dyspeptic manifestations (heartburn, belching, nausea), drugs are used that eliminate violations of gastroduodenal motility: metoclopramide (domperidone) 10 mg 2 - 3 times a day, sulpiride 100 mg IM 3 times a day.

Pain relief

If the pain syndrome is associated with damage to the parenchyma and capsule of the pancreas without involving the main duct in the process, a pronounced analgesic effect is achieved after the implementation of therapeutic measures aimed at reducing pancreatic secretion. Enzyme preparations also reduce the severity of the pain syndrome. If the pain syndrome persists, non-narcotic analgesics (analgin, baralgin) are prescribed in combination with antihistamines. In case of persistent and severe pain syndrome, narcotic analgesics can be prescribed (promedol 1 ml of 1% solution subcutaneously; morphine is contraindicated, as it causes spasm of the sphincter of Oddi) or octreotide (a synthetic analogue of somatostatin) 50-100 mcg 2 times a day subcutaneously.

With an exacerbation of uncomplicated chronic pancreatitis, the pain syndrome stops within 3 to 4 days. If within a week the intensity of the pain syndrome does not significantly decrease even with the use of narcotic analgesics, it is necessary to look for other causes of its occurrence (complications of pancreatitis, pancreatic tumor) or think about the presence of drug addiction.

Replacement therapy for exocrine pancreatic function

Mild steatorrhea, not accompanied by diarrhea and weight loss, can be corrected by

diet. An indication for the appointment of enzymes is considered to be steatorrhea with a fat loss of more than 15 g / day, combined with diarrhea and a decrease in body weight.

The enzyme preparations used should not lower the pH of gastric juice, stimulate pancreatic secretion. Therefore, the drugs of choice are considered to be enzymes that do not contain bile and extracts of the gastric mucosa. Preference should be given to microgranular forms that dissolve in the small intestine at pH 5.0 and above and mix well with food chyme. Doses of enzyme preparations depend on the degree of pancreatic insufficiency and the patient's desire to follow a diet. To ensure the normal process of digestion with adequate nutrition in patients with severe exocrine insufficiency, it is necessary to take 20,000-30000 U of lipase with each meal.

Enzyme preparations are prescribed for life, the doses can be reduced while following a strict diet with restriction of fat and protein and increased when the diet is expanded. An indicator of a properly selected dose of enzymes is stabilization or increase in body weight, cessation of diarrhea, steatorrhea and creatorrhea.

In the absence of the effect of the appointment of large doses of enzymes (30,000 U for lipase), their increase is impractical. The reasons may be concomitant diseases: microbial contamination of the duodenum, helminthic invasions of the small intestine, precipitation of bile acids and inactivation of enzymes in the duodenum as a result of a decrease in pH. At low pH of the contents of the duodenum, it is recommended to combine the intake of enzymes with antisecretory drugs (blockers of H2-histamine receptors, blockers of the "proton pump", antacids).

Spa treatment

Sanatorium treatment is carried out at the resorts of the gastroenterological profile: Essentuki, Truskavets, Jermuk, Morship, etc.

Surgery

Surgical treatment is indicated for narrowing or obstruction of the large duodenal papilla, the main pancreatic duct, choledocho-lithiasis and other reasons that impede the outflow of the gland secretion; with obstructive jaundice and an increase in the head of the pancreas; with cysts and pseudocysts of the pancreas.

Complications

Complications of chronic pancreatitis reduce the effectiveness of drug therapy and often require surgical treatment. During an exacerbation of chronic pancreatitis, hypovolemic shock, gastrointestinal bleeding, jaundice, abscesses, and sepsis may develop. Chronic pancreatitis is often complicated by pseudocysts. Long-term chronic pancreatitis leads to the expansion of the main pancreatic duct, changes in neighboring organs: narrowing of the common bile duct, duodenum, splenic vein thrombosis, effusion into the pleural, pericardial, abdominal cavity.

Forecast

Strict adherence to the diet, refusal to drink alcohol, and the adequacy of maintenance therapy significantly reduce the frequency and severity of exacerbations in 70-80% of patients. Patients with chronic alcoholic pancreatitis live up to 10 years with a complete refusal to consume alcoholic beverages. If they continue to drink alcohol, then

half of them die early. Persistent and long-term remission is possible only with regular maintenance therapy.

CHRONIC ENTERITIS

Chronic enteritis - chronic inflammatory disease of the small intestine.

According to the predominant localization of the inflammatory process, duodenitis (inflammation of the duodenum), jejunit (jejunum) and ileitis (ileum) are distinguished. More often, there is inflammation of the entire small intestine in combination with gastritis (gastroenteritis) and / or colitis (enterocolitis).

Etiology

The causes of chronic enteritis are varied. It can be caused by: 1) alimentary disorders, non-regime eating, alcoholism; 2) food allergies; 3) lamblia, helminths and other parasites; 4) chronic poisoning by some toxic chemical substances, 5) radiation injuries; 6) congenital enzymatic deficiency lesions of the small intestine. In some cases, chronic enteritis develops after repeated food poisoning.

Pathogenesisdiseases can consist of various mechanisms. The most studied: 1) direct chronic damaging effects on the wall of the small intestine (toxic, irritating, etc.); 2) immunological mechanisms: the occurrence of hypersensitivity to the products of hydrolysis of nutrients or to the products of decay of bacterial cells; 3) violation of the protective mechanisms of the intestinal mucosa; 4) dysbiosis: the small intestine, usually having a poor bacterial flora, in this disease is usually colonized by various microorganisms that are atypical for it, as a result of which digestive disorders are aggravated.

Clinical picturecharacterized by a variety of symptoms. Pains occur infrequently, are dull or spastic in nature, localized in the umbilical region. Here, it is often possible to determine pain on palpation of the abdomen and with strong pressure slightly above the navel - a symptom of Porges, pain along the mesentery of the small intestine - a symptom of Sternberg. Sometimes there is a strong rumbling and splashing during palpation of the cecum due to the rapid passage of the chyme through the small intestine, the flow of undigested and not absorbed liquid contents and intestinal gas into the cecum.

The main manifestations of chronic enteritis: 1) syndrome of enteric dyspepsia; 2) enteric scatological syndrome; 3) malabsorption syndrome.

Enteric dyspepsia syndrome manifests itself as a feeling of pressure, distention and bloating, rumbling, transfusion in the abdomen, which arise as a result of impaired digestion and absorption of nutrients in the intestine, accelerating their passage in the intestine.

Enteritic scatological syndromemanifests itself as frequent (up to 15-20 times a day) mushy, with undigested food particles, but without visible mucus, with gas bubbles in stools. Polyfecalia is characteristic, milk intolerance is often present. Exacerbations of the disease are caused by eating spicy food, overeating, food with a large amount of fat and

carbohydrates. Attention is drawn to the peculiar yellowish (golden) color of feces due to the presence of unreduced bilirubin and a large amount of fat, which gives the feces a clay appearance.

Microscopic examination of stool reveals remnants of undigested food (lienteria), drops of neutral fat (steatorrhea), crystals of fatty acids and insoluble soaps, muscle fibers (creatorrhea), free extracellular starch (amilorrhea), and a large amount of mucus.

Insufficient absorption syndrome manifested by a decrease in the patient's body weight, general weakness, malaise, and decreased working capacity.

In laboratory tests, hypoproteinemia is determined. Its presence is explained not only by impaired hydrolysis of proteins and absorption of amino acids by the intestinal wall, but also by increased exudation of proteins, mainly albumin, into the lumen of the intestine with its inflammatory lesions. The content of cholesterol in the blood is reduced. A number of specialized studies can confirm and quantify the degree of malabsorption.

Currently, many techniques have been proposed for the study of intestinal digestion and absorption. So, in the contents extracted from the jejunum with the help of a special probe, the activity of intestinal enzymes, enterokinase (norm 90-120 U / ml) and alkaline phosphatase (norm 135-300 U / ml) is determined.

The method of Ugolev is also used, the test with D-xylose. A blood test in patients with chronic enteritis often reveals anemia: either iron deficiency (due mainly to impaired iron absorption), or B12 deficiency; often anemia has a mixed character with a normal color index. The phenomena of hypovitaminosis are often observed: dry skin, angular stomatitis, hair loss, brittle nails, polyneuritis, impaired "twilight" vision. As a result of impaired absorption in the blood, the concentration of a number of ions, especially calcium, decreases. With severe enteritis, dystrophic changes are observed in the heart muscle, liver, kidneys and other organs.

The X-ray picture of chronic enteritis is more often manifested by an accelerated passage of a suspension of barium sulfate through the small intestine (sometimes the contrast mass enters the cecum within 20-30 minutes after administration, in healthy people - after 2.5 - 3 hours). Often, thickened, edematous folds of the mucous membrane are visible, in severe cases - their smoothing due to the process of atrophy.

Treatment

Prescribe a diet number 4-4a, b according to Pevzner, more stringent in the period of exacerbation and extended in the period of remission. Broad-spectrum antibiotics are not used in chronic enteritis due to the danger of increasing dysbiosis. With exacerbations of enteritis, 8-hydroxyquinoline derivatives are shown, which predominantly suppress the pathogenic intestinal flora: enteroseptol, intestopan, mexaform, mexase for 7-10 days. Digestive enzyme preparations: abomin, festal, panzinorm, 1-2 tab. or pills 3-4 times a day with meals. For the treatment of dysbiosis, colibacterin, bifidumbacterin, bifikol, lactobacterin are prescribed, containing lyophilized microorganisms that normally colonize the intestines.

During the period of exacerbation of the process, especially accompanied by diarrhea, the use of enveloping and astringent agents is shown. In severe enteritis with symptoms of insufficient absorption, hydrolysin L-103, aminocrovin and other drugs intended for parenteral nutrition are prescribed.

Of the physiotherapy procedures, the most effective are thermal, paraffin applications, ozokerite, diathermy, inductothermy. Patients are sent to the sanatoriums of Zheleznovodsk, Essentuki, Pyatigorsk, Borjomi.

Prophylaxis chronic enteritis consists in rational nutrition, prevention of toxic (domestic and industrial) intestinal lesions, timely treatment of diseases of the digestive system, prevention of secondary, concomitant enteritis.

CHRONIC colitis

Chronic colitis- inflammatory and dystrophic lesions of the colon mucosa, accompanied by impaired motor and secretory functions.

Etiology

Leading importance is attached to acute intestinal diseases, as well as invasions by protozoa, helminths. The alimentary factor is of great importance - irregular, monotonous diet, alcohol abuse, spicy foods, long-term use of sparing diets. Often, chronic colitis develops secondarily in patients with peptic ulcer, cholecystitis, gastritis, gynecological diseases (adnexitis), prostatitis, etc. The reasons may be chemical agents, some drugs, intestinal dysbiosis. The most common colitis of mixed etiology, for example, a combination of infectious and nutritional factors. Functional disorders of the intestine, primarily its dyskinesia, predispose to the development of an inflammatory process in the intestine.

Pathogenesis

An important role is played by the weakening of nonspecific immunological mechanisms, the allergic component, and dysbiosis. The previous functional disorders in the large intestine (dyskinesias, excessive mucus secretion, neuro-endocrine disorders, etc.) are of great importance.

Pathological changes in the colon are nonspecific: dystrophic changes in the epithelium of the colon, up to atrophy, expansion or atrophy of crypts, an increase in goblet cells, capillary plethora, connective tissue growths.

Clinic

The clinical picture is determined by the degree of impairment of the motor-evacuation, secretory and absorption functions of the large intestine, as well as the localization of the inflammatory process. Pain syndrome is one of the leading and persistent symptoms. Pains can be of a different nature, often aching, mainly in the lower abdomen, along the flanks, or along the colon. They can be spilled, appear and intensify with the urge to defecate and weaken after it and the passage of gas. Dull pains are often replaced by cramping pains. With the involvement of the serous membrane (perivisceritis) and regional lymph nodes (mesenteric adenitis), the pain becomes constant, increases with movement, after cleansing enemas and thermal procedures. Bloating, bloating, and stool disorders are common symptoms of chronic colitis.

The general condition, as a rule, suffers little. Minor weight loss and signs of vitamin deficiency can be associated with a sparing inadequate diet and quickly disappear with adequate nutrition.

Palpation of the abdomen reveals pain along the colon, more often in the area of the sigmoid and transverse colon. There is an alternation of spasmodic and atonic parts of the colon. The clinical examination should conclude with a digital examination of the colon to exclude hemorrhoids, swelling, abrasions, or fissures in the anal area.

Laboratory research

In the diagnosis of chronic colitis, a coprogram is necessary, sowing on bacteria of the dysentery group and dysbiosis, parasitic carriers. In the feces, you can find pus and mucus, in case of violation of the integrity of the mucous membrane, an admixture of scarlet blood. With intestinal atony, feces are compacted, with spasms - "sheep feces".

Microscopy - an increase in neutrophilic leukocytes, the appearance of erythrocytes. The presence of soluble protein in the stool indicates an inflammatory process or ulceration in the colon (positive Triboulet reaction). An increased content of organic acids (norm 14-18) indicates an increase in fermentation processes. With putrefactive processes, the amount of ammonia increases (norm 2-4). The determination of enterokinase and phosphatase helps in the assessment of enzymatic processes.

X-ray examination in chronic colitis reveals various functional disorders: atonic enlargement, spastic contractions - (rope symptom), uneven character and disappearance of haustrations, kinks, adhesions, filling defects, lengthening and expansion of the sigmoid colon. Clearer data are observed with irrigoscopy: the nature and location of the folds change, their number decreases. With increased secretion, the relief of the mucous membrane is not detected. Sigmoidoscopy is a mandatory study to exclude a tumor process and ulcerative colitis.

Differential diagnosis.

It is necessary to exclude the tumor process, dysentery, ulcerative colitis, intestinal tuberculosis. To exclude dysentery colitis and amoebiasis, it is necessary to study feces for the dysentery group and amoeba. In differential diagnosis with tuberculosis, a history of tuberculous process, predominant lesion of the ileocecal region, fever, sweating, positive tuberculin tests are important.

Mycobacterium tuberculosis in feces, as a rule, is not found.

In the differential diagnosis of chronic colitis, it is necessary to keep in mind functional disorders of the intestine, the so-called. colonic dyskinesia or irritable bowel syndrome. Dysbacteriosis occurs in all patients with chronic enterocolitis, but its severity is different, the total number of microbes changes, more often upward. The number of lactic acid sticks changes or sharply decreases, the amount of accompanying flora changes - an increase in staphylococcus, proteus, yeast, the ratio of the number of various microbes (E. coli, enterococci, clostridia) changes, microbes with altered properties appear (hemolytic forms, enteropathogenic strains of E. coli, etc.). etc.).

Treatment

As with all diseases of the digestive system, dietary nutrition is the basis. It is recommended to use diet number 4, in addition, it is necessary to take into account the properties of various products to influence the function of bowel emptying.

Etiotropic treatment consists in influencing the etiological factors of colitis, or their elimination. Unfortunately, this is not always possible. It is necessary, strictly according to indications, to take those medications that can lead to damage to the colon. If a bacterial pathogen is identified, antibacterial and antiparasitic agents are prescribed.

Pathogenetic therapy includes measures aimed at eliminating dysbiosis, the effect on impaired intestinal motility; physiotherapeutic and sanatorium-resort treatment can also be attributed to the methods of pathogenetic therapy.

Correction of dyskinetic disorders directly causing symptoms such as pain and stool disturbances is one of the most difficult tasks, because most patients develop dyskinesia,

i.e. multidirectional motility disorders in different parts of the colon. Therefore, the use of anticholinergic and adrenergic drugs does not always give a positive effect. Nevertheless, if clinically it is possible to assume the predominance of the spastic component, a good effect is achieved with the use of atropine and its derivatives, antispasmodics: (no-shpa, fenikaberan).

Pathogenetically more justified in dyskinesias is the appointment of enzyme preparations, which, as shown by clinical experience, have an analgesic effect in patients and normalize the stool. For fermentative dyspepsia, drugs are prescribed, which include amylase and hemicellulase, which catalyze the hydrolysis of cellulose (enzistal, festal, digestal, etc.), and in cases where putrefactive dyspepsia predominates, drugs with sufficient proteolytic activity (panzinorm, pancitrate, creon, festal) are prescribed.

Physical factors have a beneficial effect due to the influence of paskinetic disorders. The effectiveness of inductothermy, sinusoidal modulated currents, magnetotherapy has been reported. Spa treatment is effective in the remission phase: Goryachy Klyuch, Essentuki, Zheleznovodsk, Karmadon, Nalchik, Sestroretsk, Staraya Russa. The main curative factors in the resorts are mineral waters and curative mud.

FUNCTIONAL DISEASES OF THE INTESTINAL

The actual problem of modern medicine is the so-called functional diseases, i.e. diseases in which conventional research methods, including X-ray, endoscopic, light microscopy of biopsy specimens, do not reveal an organic, morphological substrate.

About the causes and mechanisms of IBS developmentthere is no consensus. In IBS, it was not possible to identify motility disorders specific to these patients. The study of the characteristics of pain perception and cerebroenteric relationships turned out to be more successful. Studies using the balloon-dilation test reveal in patients with IBS a decrease in the threshold of rectal pain sensitivity during rapid mechanical stretching of the rectal wall. Moreover, in IBS, a diffuse pain perception impairment was revealed throughout the intestine.

The characteristic length of the SRK is the presence of abdominal pain in combination with diarrhea or constipation.

Abdominal pain can be of varying intensity, localized, as a rule, in the lower abdomen, although it can also be noted in other parts of it. It often intensifies after a violation of the diet, with a surge of emotions, against the background of nervous and physical fatigue. The pain usually decreases after the act of defecation or after the passage of gas and, which is very important, does not bother at night. Along with pain, patients are usually worried about a change in stool frequency: stool is considered abnormal more than three times a day or less than three times a week. There may be a change in the shape and consistency of feces, the appearance of mucus in the feces. Patients may present complaints characteristic of neurocirculatory dystonia with various syndromes - cardialgic, asthenic, cephalgic, etc.

A distinctive feature of IBS is the variety of complaints - both gastroenterological and non-gastroenterological, as well as the presence of neuropsychiatric disorders. A long course of the disease and resistance to treatment with purely gastroenterological agents are considered typical. With relapses of the disease, a connection between exacerbations of the disease and psychoemotional stress is usually seen.

The pains are very diverse, often sharp, cramping, aggravated by emotions, immediately after eating (gastro-cecal reflex), a few hours after eating food containing fats, smoked meats. Patients note relief of pain after stool, discharge of gas, less often they complain of dull, aching pains. Attention is drawn to the discrepancy between a large

number of complaints, a long course of the disease and a satisfactory general condition, good appearance, and the absence of objective changes in the status of patients.

An endoscopic examination of the colon is decisive in the diagnosis. With IBS, no pathological changes are found in the mucous membrane. At the same time, violations of the bowel tone, spasm, especially in the distal part, are often found. Rectoscopy, sigmoidoscopy, colonoscopy are poorly tolerated by patients with IBS. The procedure is accompanied by sharp soreness, the introduction of the drug, the injection of air causes pain in the patient, similar to those stated in the main complaints.

A common understanding of the IBS is facilitated by the indicators carried out in the "Rome criteria" and presented below.

The presence of two or more of the following symptoms for 25% of the duration of the disease:

- Pain and discomfort in the abdomen:

- pass after the act of defecation;

- associated with a change in stool frequency (constipation, diarrhea, or their alternation);

- associated with a change in the consistency of feces.

- Changes in stool frequency (more than 3 times a day or less than 3 times a week).
- Change in the consistency of feces (liquid, solid).
- Change in the act of defecation.
- Imperative urge.
- Incomplete bowel emptying.
- Extra effort during bowel movements.
- Excretion of mucus in the feces.
- Bloating, rumbling, flatulence.

A diagnosis of IBS is a diagnosis of exclusion. Similar complaints can be presented by a patient with a prognostically unfavorable organic pathology, only by excluding which, the doctor can stop at the diagnosis of a functional disease.

It is very important to highlight the criteria for excluding IBS.

The criteria for excluding the diagnosis of IBS (symptoms of "anxiety") include:

-unmotivated weight loss;

-the presence of symptoms at night;

-the onset of the disease in old age;

-fever;

-the presence of blood in the feces;

-leukocytosis;

-anemia;

-acceleration of ESR;

-changes in the biochemical blood test.

Endoscopic examination, supplemented by biopsy if necessary, is of decisive importance in the diagnosis. Irrigoscopy allows you to determine the presence and nature of organic pathology of the colon, as well as to identify a violation of motor function. Stool microscopy makes sure that there are no erythrocytes and leukocytes, demonstrates the presence of mucus and often iodophilic flora.

It is believed that the somatic, psychiatric and social prognosis in patients with

functional impairments is unequal.

The somatic prognosis is generally favorable, since even with a long course of the disease, there are no pronounced organic changes in organs and systems. While somatic symptoms are usually reduced under the influence of treatment, neurotic manifestations usually persist.

Diagnosis and treatment of patients should be carried out in a well-organized interdisciplinary relationship. Coordination of the actions of the therapist, gastroenterologist and psychiatrist (specialist in borderline mental disorders) is an essential condition for successful therapy.

Treatment includes psychosocial adjustment measures, diet therapy and drug therapy depending on the prevalence of symptoms - pain, diarrhea and constipation.

It is very important to correctly assess the mental state of a patient with IBS. It is necessary to focus the patient's attention on the normal indicators of research and constantly emphasize the importance of the absence of pathological disorders for the prognosis of the disease. The patient must believe in the absence of a serious, life-threatening organic disease. Such activities largely determine in which group the patient will be - in the group of “patients” or “non-patients” of IBS, which greatly affects the effectiveness of subsequent therapy.

Smoked meats, peppers, vinegar, strong tea and coffee, alcohol, and foods that cause excessive gas formation are excluded from the diet of patients. Patients with a predominance of constipation are offered a diet rich in fiber (bread,carrots, beets, apples, etc.), as well as small white wheat bran up to 30 g / day. For many non-patients, psychosocial support and dietary interventions are sufficient in combination with short courses of drug therapy.

There are still no medications that are certainly effective for IBS. Most often, antispasmodics are used to relieve abdominal pain and reduce flatulence. Myotropic antispasmodics, selective antagonists of calcium channels dicetel and mebeverin have significant efficacy in pain syndrome. Their advantage is a selective effect on the smooth muscles of the intestine and the sphincter of Oddi, as well as the absence of side effects characteristic of anticholinergic drugs.

Many authors consider it necessary to include tranquilizers and antidepressants in the complex therapy of IBS. In case of constipation, if the enrichment of the diet with ballast substances is insufficient, they resort to the appointment of osmotic laxatives: lactulose, magnesia milk and macrogol (forlax). The setting of cleansing enemas should be avoided, since, by mechanically stimulating the parasympathetic receptor apparatus of the distal colon, they increase the pressure in its cavity, which can provoke spasm, pain and increased secretion. Imodium (loperamide hydrochloride) is used to treat diarrhea in patients with IBS. It suppresses the rapid propulsive contractions of the intestine, which leads to a slowdown in the movement of feces. This is accompanied by a decrease in the passage of the liquid part of the chyme, helps to increase the absorption of fluids and electrolytes. In addition, the drug reduces the susceptibility of the colon wall to stretching, which increases the pain perception threshold, softens and eliminates tenesmus. With the dominance of diarrhea in the clinical picture of IBS, smecta can be used, as well as enveloping agents - bismuth nitrate.

The most effective in IBS is complex therapy with the inclusion of all the necessary tools and methods, provided that each of them occupies an adequate place. Of particular importance for the treatment of IBS patients is the constant adherence to the principle of an individual approach to each specific patient.